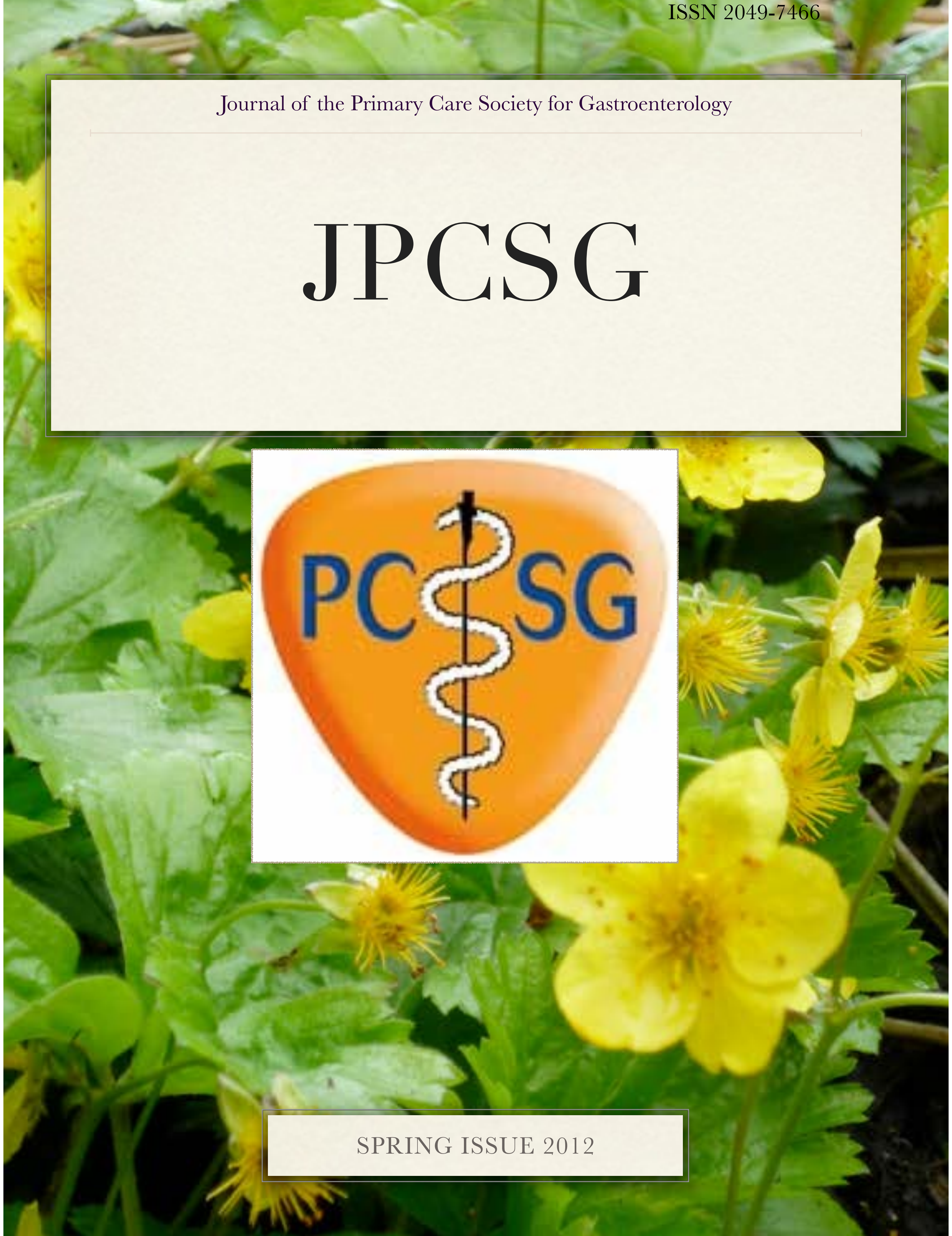
ISSN 2049-7466

Journal of the Primary Care Society for Gastroenterology

JPCSG



SPRING ISSUE 2012

Journal of the Primary Care Society for Gastroenterology

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CONTENTS

Editorial

Welcome to the the Spring Issue of the JPCSG.In these days of torrential rain, I hope this issue brings a glimmer of academic sunshine.

This issue is heavily based on case histories. Case histories are often overlooked as a teaching method but this is unfortunate as they can reveal not only important lessons but one can learn from the thought processes of the clinician involved.

I have also added an article penned by my good self on the UK IBD Audit that the PCSG has been a part of .

We have an exciting Annual Scientific Meeting in October with talks on paediatric and tropical gastroenterology, end of life issues in Liver disease and the emotional and psychological issues seen in gastrointestinal illnesses.

Again, the PCSG would like to thanks for our sponsors for their continued support and you, our wonderful members for all you do. We hope to see you all at one or even all of our meetings in 2012.

John O’Malley, Editor.

INTRODUCTION

- *IBD is often seen, rightly in some respects, as a rare illness and the treatment of exacerbations an unusual reason to see a patient. But taken as a whole, IBD is not a minor problem and the total cost to the NHS has been estimated at £720 million with an average cost of £3000 a patient per year. The UK IBD audit has, until recently, mainly centred on the secondary care aspect of IBD care allowing secondary care providers the ability to benchmark their services against others.*

SECTION 2

The UK IBD Audit; the primary care questionnaire

What is the role of the primary care in the care of IBD patients?

John O'Malley, Secretary PCSG

What should be the role for primary care in the care of patients with IBD?

The perception of many is that IBD is a rare illness best seen in secondary care. However, with the same voice many are also saying that well controlled colitics and those with stable Crohn's Disease should be put back in the community for ongoing monitoring and even treatment of flare ups. The cost implications are a major driving force for this but the principle could also free up outpatient space for patients who require treatment for flare ups and those newly diagnosed. But is primary care ready for this new way of working?

The UK IBD audit is a joint working group comprising of representatives from various organisations such as the BSG, BSPGHAN, the RCP and Crohn's and Colitis UK. It was commissioned by the Healthcare Quality Improvement Partnership and Healthcare Improvement Scotland.

The Audit has gradually grown through its lifetime to include assessment of paediatric care and now in its present form, primary care.

But why IBD? IBD although uncommon in individual practices is a costly illness. The total cost to the NHS for the care of patients with IBD has been estimated at £720 million based on an average cost of £3000 per patient per year. Half of these costs are attributable to the care of patients who relapse.

However, as with many illnesses, it became obvious from the 1st audit that there was a wide variation in both access and quality of care and a more rounded view was required. To complete the picture, paediatric care was added in the second round followed by the inclusion of primary care and inpatient questionnaires in the 3rd.

How was the data collected? Following admission, a primary care questionnaire would be sent out to the named GP and the replies collated online or else the via the freepost envelope supplied. Data from a total of 1675 questionnaires were collected and analysed.

The results

59.5% of GPs reporting seeing the patient in the 4 weeks previous to admission with the likelihood of those patients having a non elective admission being seen far greater. What that means for the other 40.5% is open to debate. It could mean they 'self managed' their admission and were able to get in touch with the hospital IBD nurse or similar about their relapse. It could also suggest that some patients had little faith, either through experience or not, that their GP could handle their flare up. Children, as a group, were far less likely to have seen their GP prior to admission.

If the GP did see them , what did they do? In a third of cases, both adult and paediatric, the GP neither contacted secondary care or initiated treatment. So what was the point of the consultation? Did this non intervention lead to admission? Treatment was more likely to happen if the GP saw the patient more than once (44% in adults and 25% in children). Confidence in treating IBD showed some interesting features. GPs seemed much happier in treating UC than Crohn's and the favoured treatments tended to be either raising the dose of the 5 ASA medication or else commencing steroids.

I think we can all wish for better communication between secondary and primary care and it was obvious that it needed to be improved in IBD. 59% of GPs reported having no contact with secondary care in the 4 weeks prior to the patient's admission. When they did make contact it tended to be aconsultant in 81% of cases with the SpR in 11% and the nurse specialist in 17%.

The poor use of IBD nurses was also highlighted. Where a hospital did have a IBD nurse available, primary care only contacted them in 9.3% of cases in adults and 8.6% in children(Tables 1 and 2).

Table 1

ADULT PATIENTS	
	Where IBD nurse provision was available
All adult patients	
Contact made with IBD CNS	Yes=99 (9.3%)
UC adult patients	
Contact made with IBD CNS	Yes= 56(10.7%)
CD Adult patients	
Contact made with IBD CNS	Yes=43 (8.0%)

Table 2

PAEDIATRIC PATIENTS	
	Where IBD nurse provision was available
All paediatric patients	
Contact made with IBD CNS	Yes=10 (8.6%)
UC paediatric patients	
Contact made with IBD CNS	Yes=3(8.3%)
CD paediatric patients	
Contact made with IBD CNS	Yes=7 (8.6%)

It was encouraging to see that, where there was IBD nurse provision, the GPs were slightly less likely to contact the consultant and slightly more likely to contact the IBD nurse. This suggests promotion of the work of specialist nurses by secondary care should be a priority. Also advice was more likely to result in prompt treatment from GPs who did get in contact.

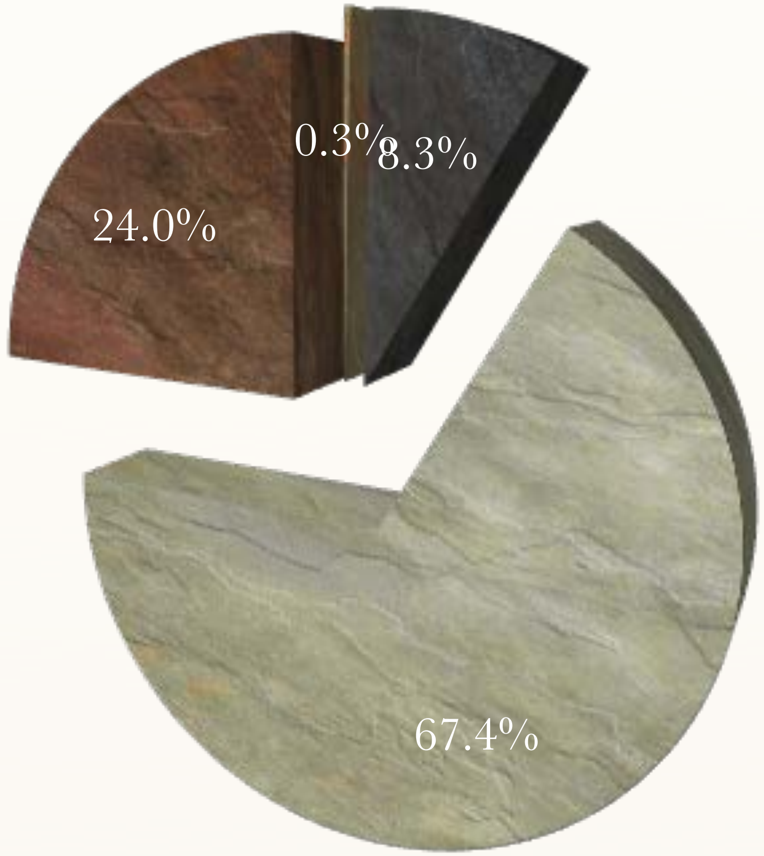


Table 3

Confidence in diagnostic ability.

An important aspect, especially in this era of GP commissioning , was whether GPs felt confident enough in recognising the key symptoms of IBD.

Most felt confident, to a varying degree, as to their ability to diagnose an exacerbation (8.3% slightly confident, somewhat confident 67.3% and very confident 24%) with only 0.3% not feeling confident.

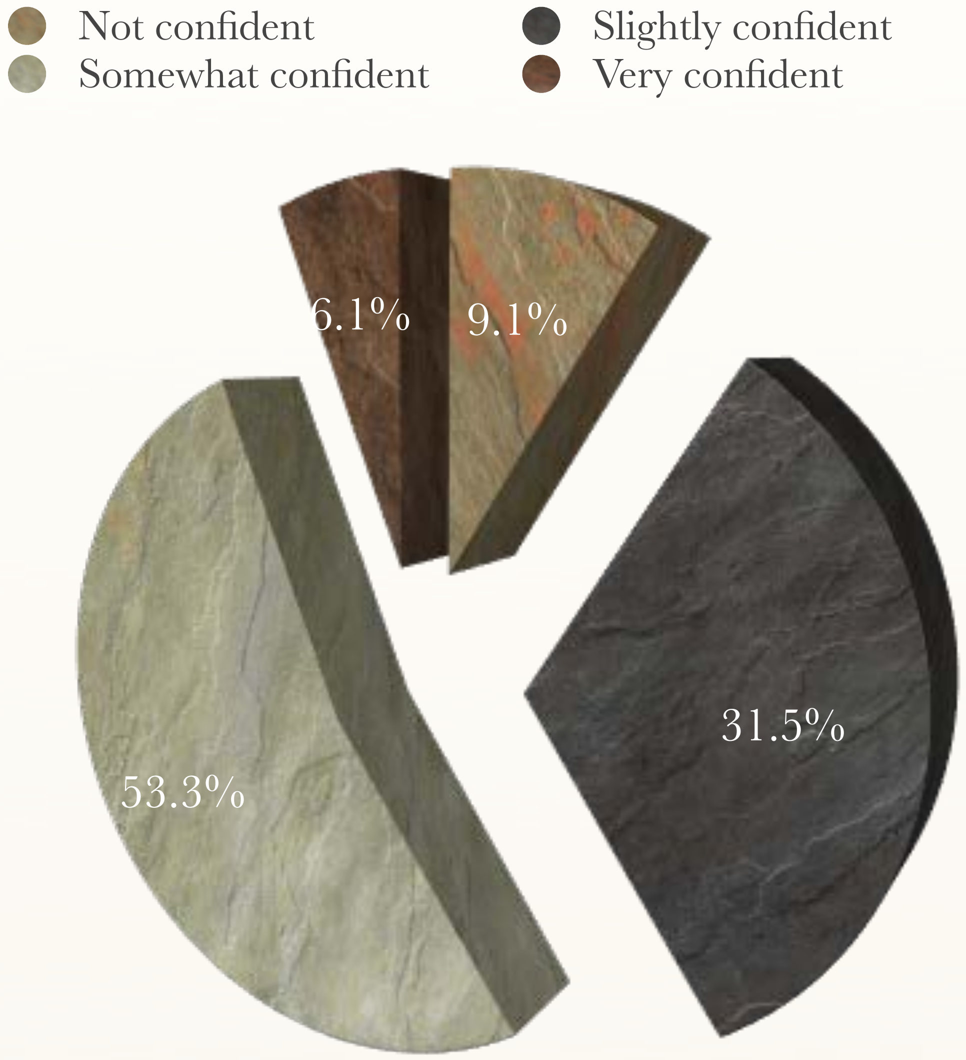


Table 4

Confidence in treatment of exacerbations.

Levels in treating IBD exacerbations showed greater numbers being not confident at all with levels of 9.1% noted (slightly confident 31.5%, somewhat confident 53.3% and very confident 6.1%).

When taken together with other data, the results suggested that there was a link between confidence in both aspects cited and whether those GPs had easier access to secondary care advice. It was of note that those who felt the least confident were the most vocal in wishing for increased educational support and those who felt the most confident were usually the ones who knew who to contact for advice.

So how easy is it to contact the hospital and get an urgent appointment? In 59% of cases, GPs said they did not have access to an outpatient appointment within 7 days. What was very interesting was that the audit was able to identify the hospitals used and then compare it to the results of the hospital audit. Looking at the hospitals cited in this part of the study, 86% of IBD services stated that they could offer an appointment for IBD patients with flare ups within 7 days. Whatever the hospitals think they are offering, it is obvious that the GPs perceive the actual situation to be different.

Finally, two questions looked at patients under exclusive primary care follow up. In patients with total colitis, the risk of cancer is markedly raised yet with a well organised surveillance programme, the risk can be less than the general population. As many CCGs are now pushing to increase the numbers of IBD patients seen in primary care, it is important and I, personally, would say ethically paramount, to have protocols in place which emulate that seen in secondary care. It was, therefore, worrying to see that the figures suggest many patients may not be getting the surveillance thy need. When asked whether GPs knew what were the colon cancer surveillance requirements of their patients (wholly seen in primary care) 2.5% said yes, 18.2% said no and 15% didn't know. That left 64.3% who said the question was non applicable. Why? Was this because they didn't know? Even more worrying was that in the 2.5% where the colon cancer surveillance needs were known only 63.9% were being screened with 16.7% not and a further 19.4% where it was unclear as to whether they were being screened or not.

Conclusion

This audit completes the circle of care analysis between secondary and primary care and raises many issues but also lots of further questions. What is happening to patients who are not being followed up by anybody? With the need to cut costs, CCGs are pushing for 'well controlled' IBD patients to be moved back into the community. The question remains is the community ready for them.

Key Recommendations

- Increase use and awareness of IBD nurses, promoting the valuable role and support they can offer.
- An up to date management plan should be given to GPs for each patient with 'who to contact' details.
- The PCSG has a valuable part to play in improving education.
- Primary care, with the help of the PCSG, should encourage research into the numbers and levels of care given to IBD patient not seen in secondary care or actively by primary care.
- GPs require educational support in relation to the colon cancer surveillance requirements of patients with IBD.

The full report can be found at;

www.rcplondon.ac.uk/sites/default/files/hqip-uk_ibd_audit-primary_care_questionnaire_report-12_april_2012.pdf

INTRODUCTION

- *My thanks to Mike Cohen for highlighting the following two cases which lead on nicely (Ah, the editorship of Vanity Fair awaits) from the last article. IBD, like many illnesses, refuses to read textbooks and thus can present in ways which are not typical. Mike highlights two cases where fever was the predominant symptom of flare ups.*

SECTION 3

Fever and exacerbations of IBD

Mike Cohen, GP and Endoscopist, Bristol

The 2 cases illustrate fever as a presenting symptom during an exacerbation of IBD.

Case1

A 57 year old man presented with proctitis 20 years ago. Originally he was treated with sulphasalazine tablets and Predsol enemas. He had been well without exacerbations for nearly ten years and was currently was not taking any treatment. At beginning of December 2010 he developed some diarrhoea and was opening his bowels 4-6 times/day. He had originally been followed up in hospital but he strongly indicated he preferred not to go to hospital this time if possible. He is needle phobic and has always been reluctant to have blood tests. He was restarted on sulphasalazine 1g bd which was increased after one week to 1.5g bd when it failed to improve his symptoms. I first saw him at this time and prescribed Asacol suppositories

500mg bd. He did not get on well with this as he said he could not retain them. He saw our nurse practitioner who changed him to oral mesalazine 400mg tds and urged him to have blood tests which had previously been recommended

One week later he presented with a fever and pain over his right face. There was right sided parotid tenderness. His diarrhoea was improved. He agreed to having blood tests and had FBC CRP and LFT done. His mesalazine was stopped in case these symptoms were due to an adverse drug reaction.

Over the next few days his diarrhoea recurred. Consequently he was started on prednisolone 30mg daily.

Bloods showed:-

Hb 11.3 (NR 13-17)

White cell count 9.1 (NR 4-11)

Platelets 628 (NR 150-450)

CRP 89 (NR<5.0)

Albumin 24 (NR 35-50)

Alkaline Phosphatase 29 (NR40-130).

Stool culture was negative and C Difficile toxin was negative

I reviewed him at home one month after he originally presented. He was completely washed out and had lost 2 stone in weight. He reported marked night sweats. He agreed to a gastroenterology referral which was arranged. Over the next 4 weeks he remained unwell with an intermittent fever and night sweats. His bowels had settled and his steroids were being reduced by 5mg per week. He had been seen in clinic and his consultant noted his abnormal bloods and commented "*it is unusual to develop more extensive colitis at this late stage and it would be difficult to explain all his symptoms on proctitis as non intestinal manifestations are unusual with limited colonic disease*".He wondered whether the patient may have a lymphoma or viral infection. A CT scan of the abdomen and pelvis was organised.

This was performed on 21 February 2011 and was reported as showing extensive if not total colitis. No liver or biliary tract abnormalites were seen. Only oral contrast was used as the patient declined IV cannulation.

MRCP was normal.

He was visited at home on 25 February 2011. He was very weak and unable to get out of bed. He had drenching night sweats. Bowels were now open 5/day and 5/night and his pulse was 130/m with a BP of 110/60. Examination of his abdomen showed that it was soft but distended. An emergency admission was arranged. The patient was treated with IV hydrocortisone and oral steroids. Pentasa 2g/day was added. There were no adverse effects to this medication.

He was discharged on 3rd March 2011 and has steadily improved. He has however not yet been reviewed in gastroenterology clinic. The patient had cancelled follow up appointments on 24th August and 30th September 2011. He remains on Penatasa MR 2g daily.

Comments

In retrospect this man clearly had an exacerbation of his inflammatory bowel disease. His management was hindered by his reluctance to have blood tests and a wish to be treated at home.

Review of literature shows that sulphasalazine can cause parotitis and this must have been the cause of his right sided facial pain.

Fever is a well recognised symptom associated with exacerbations of IBD. However its importance is not well emphasised in more recent textbooks. The Truelove and Witts criteria from 1955 grade colitis into mild, severe or fulminant attacks. The presence of fever is an important discriminating factor for identifying the severity of an attack.

Interestingly his follow up has also been delayed seemingly due to his reluctance to engage with the clinic.

Case 2.

A 34 year old GP with known Crohn's Disease presented with symptoms of fever and night sweats over the past four weeks. She also complained of some generalised arthralgia and backache. She looked well and examination of her abdomen was normal.

Hb 12.9,

WBC 5600,

Viscosity 1.57

CRP 20.9.

I suspected intra-abdominal sepsis associated with her Crohn's Disease and referred her urgently to her gastroenterologist. She was seen the next day by the IBD nurse who arranged an MRI of her small bowel:-

The appearances are of active inflammation of the caecal pole but no definite small bowel involvement is seen. No intra-abdominal collections are identified.

Viral blood screen was negative and she had a normal CXR. She had been taking Pentasa 2g bd. Subsequently she was started on a reducing dose of prednisolone and quickly improved. Adalimubab injections were then commenced and I am pleased to say she has stayed in remission for the last 4 months

Comments

This patient is well informed and aware of possible symptoms that may mean her having an exacerbation of her disease. I was concerned that she may be developing a possible intra abdominal abscess associated with her IBD but in fact the fever resulted from an exacerbation of her IBD alone.

My referral was dealt with very promptly and the necessary investigations arranged by her IBD nurse in conjunction with her consultant.

She has responded well to this new biological agent and is monitored regularly in secondary care.

Editor’s note

Fever is often not assessed,even in clinics, during flare ups and can often be low grade and fluctuating. Even without flare ups, it still can be a significant symptom if looked for especially in Crohn’s Disease.

A study of patients in Canada showed that in any 3 month period patients with Crohn’s Disease as compared to UC were more likely to complain of fever or night sweats (24% compared to 15%)[1].

[1] *Singh S, Blanchard A, Walker JR, Graff LA, Miller N, Bernstein CN. Common symptoms and stressors among individuals with inflammatory bowel diseases. Clin Gastroenterol Hepatol 2011;9(9): 769-75.*

INTRODUCTION

- *Adeel Saleem in this case history highlights how complicated an illness Ulcerative Colitis is and how its extra intestinal features often can have serious consequences. Treatment of primary sclerosing cholangitis and the use of transplantation has meant that malignancy remains the major cause of death.*

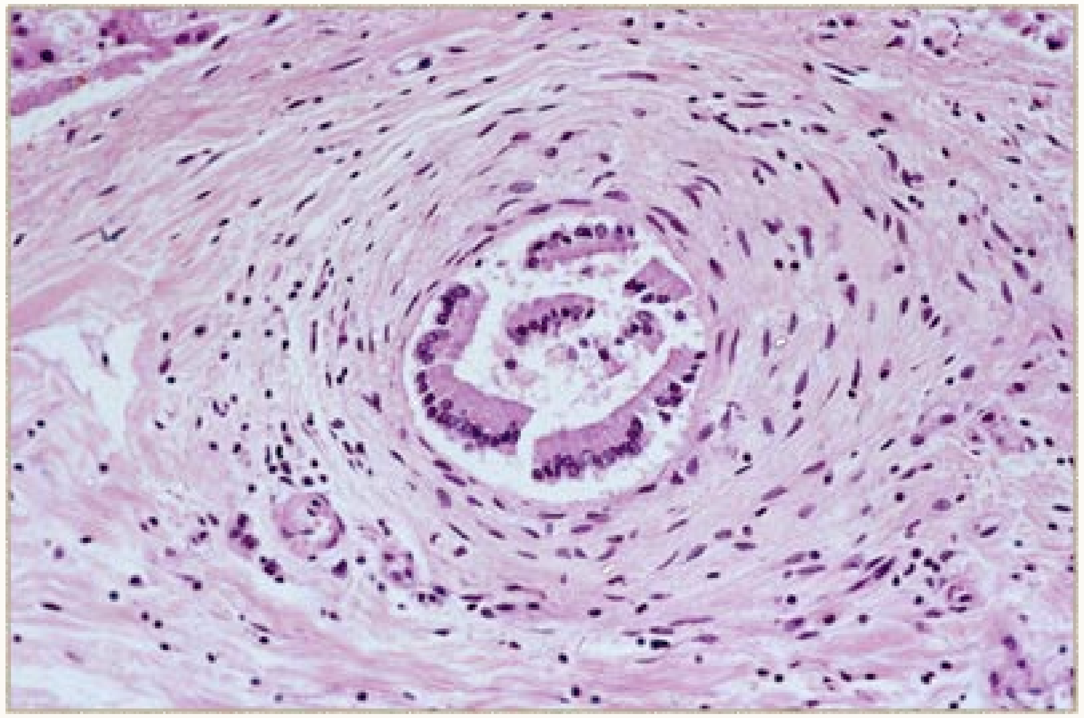
Primary Sclerosing Cholangitis

Adeel Saleem, GP and endoscopist, Edinburgh.

A 52 year old female with a known history of Ulcerative colitis for 18 years presented with fatigue, bruising and intermittent abdominal pain. She had had a total colectomy with an ileo-anal pouch replacement about 10 years ago mainly due to poor response to medical treatment. Since then her condition had been in complete remission and she hadn't had any gastroenterologist follow up for many years. She had blood tests for fatigue, bruising & intermittent epigastric pain.

Her blood tests showed slightly deranged LFTs with ALT 76, Alkaline phosphatase 166 and GGT of 152. About one month later her ALT increased to 119 and Alkaline phosphatase remained high but with no significant change. Her other significant medical history was a previous thyroid carcinoma treated by a total thyroidectomy. She remained on thyroxine 125mcg daily. All her other investigations including hepatitis serology, HIV, U&Es, caeruloplasmin, TFTs, iron studies, auto antibody screen and clotting profile were normal.

An MRI scan of the abdomen confirmed a diagnosis of sclerosing cholangitis. It showed extensive irregularity & ectasia of intra-hepatic and extra hepatic biliary tree with thickening of neck of gall bladder. A liver biopsy was arranged as well.



The histology shows the classical changes of periductal 'onion skin' fibrosis and inflammation. There is also signs of portal oedema and bile duct proliferation which leads to expansion of the portal tracts.

Discussion:

Primary sclerosing cholangitis is a rare disease of unknown aetiology characterised by chronic inflammation and fibrosis of the bile duct. Narrowing of the bile duct lumen usually occurs throughout the biliary tree. Less commonly, changes are confined to the intra-hepatic or extra-hepatic ducts but are seldom short enough to resemble a post-traumatic or focal malignant stricture.

- There is a strong association with inflammatory bowel disease (75 per cent of cases), particularly ulcerative colitis but also Crohn's disease.
- In addition to complications related to end-stage liver disease, patients may develop a number of other complications e.g. biliary strictures, infective cholangitis, cholangiocarcinoma.
- The majority of patients will be asymptomatic at the time of diagnosis, although they may have advanced disease.

The diagnosis of primary sclerosing cholangitis should be suspected in a patient with ulcerative colitis who has abnormal LFTs, especially a raised alkaline phosphatase. Pruritus and fatigue are early symptoms and patients may also present with fevers, night sweats and right upper quadrant pain (1).

Other features:

- hypergammaglobulinaemia occurs in one third; IgM levels increased in 50% of cases
- raised hepatic copper levels in nearly 90% of cases
- auto antibodies are less frequent than in autoimmune chronic active hepatitis and primary biliary cirrhosis:
- smooth muscle antibodies occur in 11% of cases

- P-ANCA positive in more than 50% of cases
- antinuclear antibodies in 6 to 35% of cases
- antimitochondrial antibodies almost never occur

Liver biopsy may reveal:

- a fibrous obliterating cholangitis
- loss of interlobular and adjacent septal bile ducts

ERCP may demonstrate:

- multiple annular strictures, separated by round or slightly dilated duct segments,
- the intrahepatic and extrahepatic bile ducts showing a beaded appearance

- Ursodeoxycholic acid may improve liver function and the patient's symptoms but no definitive improvement in histology or mortality or delay in liver transplant has been demonstrated (1).

Other measures include:

- cholestyramine to relieve pruritus
- correction of deficiencies of fat-soluble vitamins
- percutaneous transhepatic balloon dilatation may be of benefit for dominant strictures

Liver transplantation is potentially curative and is indicated where there is hepatic failure, ascites, or oesophageal varices. A 70% five-year survival is reported in major transplant centres but the timing of transplant and patient selection are critical. PSC recurrence in the newly transplanted liver has not so far been reported. Colectomy for concurrent ulcerative colitis does not affect the outcome of liver damage.

It should also be noted that about 20 per cent of patients will develop a dominant biliary stricture so it is important to exclude a cholangiocarcinoma (which is common) (1).

The mean survival for all patients is about 7 years, rising to 8-15 years for asymptomatic individuals. About 10% develop cholangiocarcinoma.

Patients usually die from complications related to:

- secondary biliary cirrhosis
- portal hypertension
- cholangitis

Patients who were asymptomatic at the time of diagnosis have a better long-term survival.

There is no relation between the course of the disease and that of accompanying inflammatory bowel disease. PSC often occurs and worsens in patients whose inflammatory bowel disease has become quiescent after.

Reference:

1. Prescriber 2003; 14(23):20-9.

<u>Editor's note</u>

The importance of looking for malignancies in PSC cannot be overemphasised. In Fevery's recent study of 200 patients with PSC, malignancies developed in 40 patients and led to death in 28. The main cancers were cholangiosarcoma and colorectal carcinoma[1].

IBD with PSC does seem to be a distinct type of colitis in which low endoscopic activity often belies an active histologic inflammation that may increase the risk of malignancy. Furthermore, liver transplantation seems to have an effect on colonoic inflammation is such types[2].

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1 Fevery J, Henckaerts L, Van Oirbeek R, Vermeire S, Rutgeerts P, Nevens F, et al. Malignancies and mortality in 200 patients with primary sclerosering cholangitis: a long-term single-centre study. Liver Int 2012;32(2):214-22.

2 Jorgensen KK, Grzyb K, Lundin KE, Clausen OP, Aamodt G, Schrumpf E, et al. Inflammatory bowel disease in patients with primary sclerosing cholangitis: clinical characterization in liver transplanted and nontransplanted patients. Inflamm Bowel Dis 2012;18(3):536-45.

INTRODUCTION

- *Life does not share out illness in a fair way and it is important to remember that often, two or even more illnesses may explain the symptoms. I have often seen patients who have had their rectal bleeding explained away because of haemorrhoids and the concurrent tumour has been missed. Dorothy King highlights such a case where not everything is what it seems.*

SECTION 5

What you see is not necessarily all you get.

Dorothy King, GP. Chester.

Patient FB female aged 42

I present this case because of the excitement it generated among my consultant colleagues, who were exercised by the concurrent diagnosis of two separate gastrointestinal conditions.

The patient presented describing symptoms which seemed to justify screening for coeliac disease as well as referral for sigmoidoscopy, so it seemed appropriate to investigate on that basis.

First presentation with bowel symptoms (to a GP colleague):

September 2009

FB reported a change in bowel habit for 2 months, from a regular normal stool passed daily to alternating constipation & loose stool.

She also described fresh rectal bleeding for a few days – initially fresh red loss, then becoming mixed with stool and associated with larger amounts of darker red loss.

She was referred privately for a lower GI surgical opinion. Colonoscopy and biopsies were normal, including histology.

She was advised to increase fluid intake & use Fybogel, and her symptoms resolved.

New presentation:

October 2011

FB returned with a 6 week history of increased frequency (up to 4 times a day) of loose stools with blood & mucus.

Abdominal & rectal examinations were normal

BMI 21

FBC, ESR, CRP, U&E, LFT, TFT normal

Stool MC&S: no infection

Transglutaminase antibodies 14.3

Further investigations:

Fast track referral for lower GI investigation

Open access OGD referral for D3 biopsies.

Subsequent flexible sigmoidoscopy revealed colitis and the histology was reported as suggestive of Ulcerative Colitis

The colorectal surgeons referred the patient to the gastroenterology physicians for management and follow up, with no immediate treatment provided.

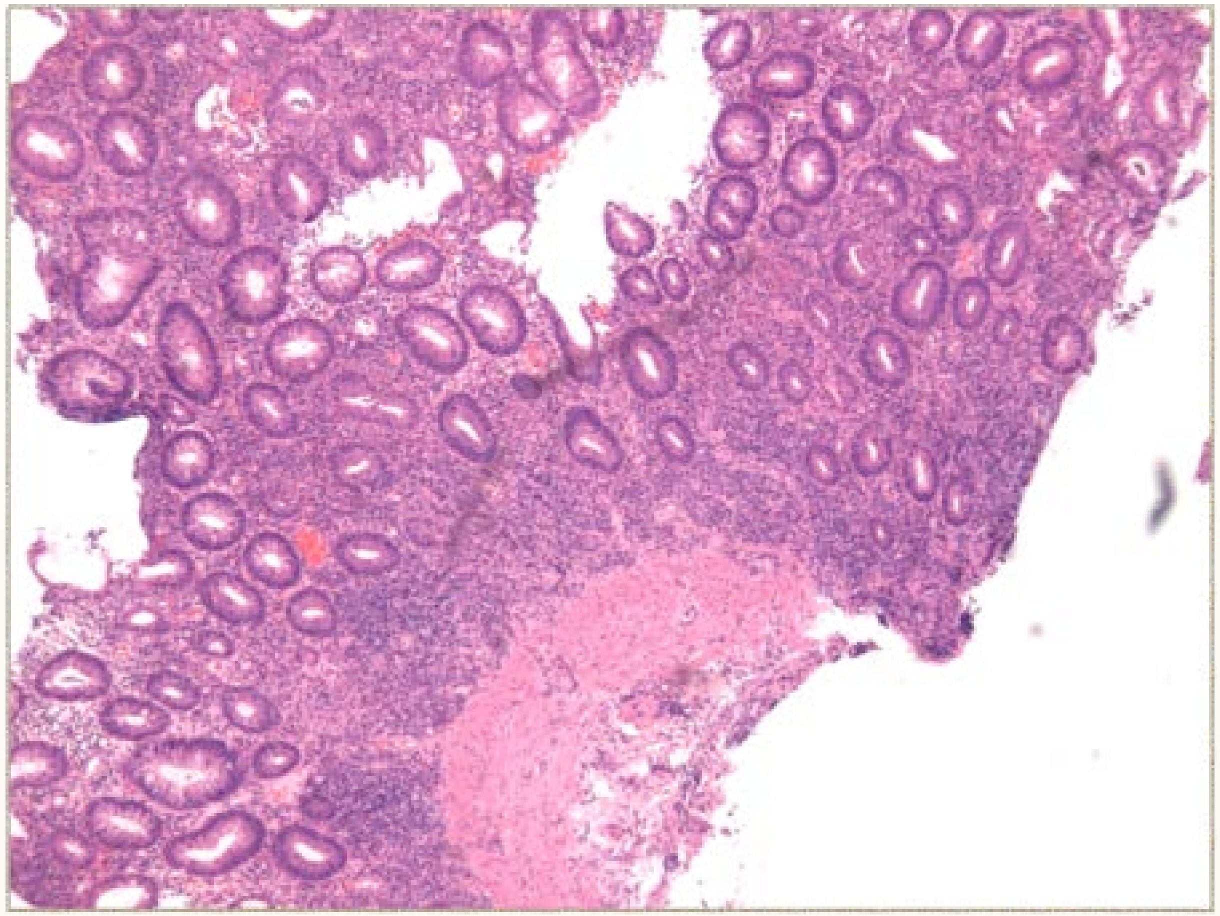
The patient attended her GP as the symptoms persisted.

Initial Rx: Mezavant XL 2 daily + Mezavant enemas

Oral therapy caused nausea but the enemas were continued, with complete resolution of bleeding and pain.

The patient had health insurance so was referred privately to a gastroenterology physician.

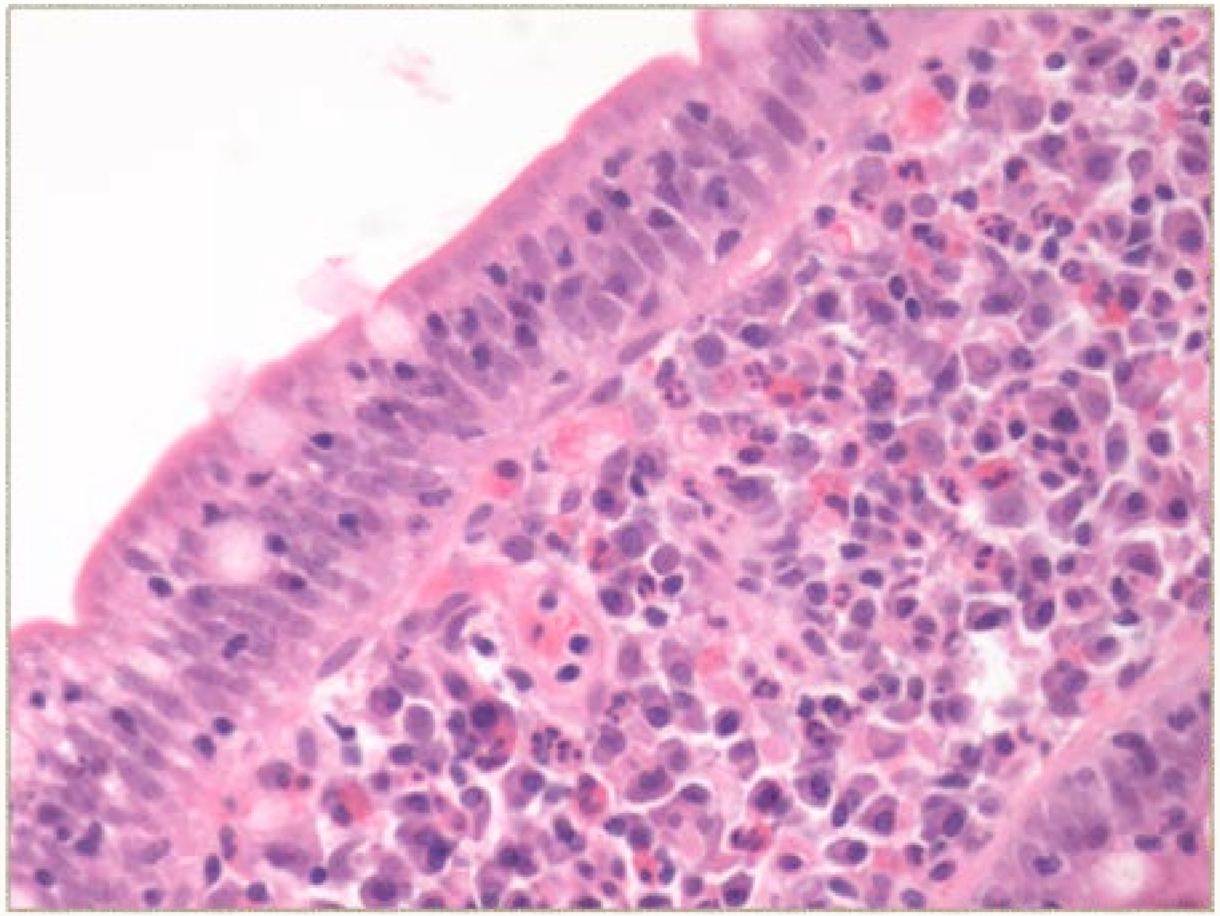
At OGD D3 biopsies confirmed coeliac disease



Slide 1Duodenal biopsy at high power showing increase in intraepithelial lymphocytes and inflammation of lamina propria.

The gastroenterologist requested review at the histology MDT meeting to confirm that these were separate diagnoses rather than microscopic colitis affecting much of the bowel.

The large bowel histology showed severe chronic inflammation, goblet cell distortion and mucin depletion in keeping with chronic inflammatory bowel disease (IBD) suggestive but not totally diagnostic of ulcerative colitis (UC).



Slide 2 Large bowel biopsy showing severe chronic inflammation, goblet cell distortion and mucin depletion in keeping with chronic inflammatory bowel disease.

It was agreed to diagnose IBD pending the outcome of full colonoscopy. It was not possible to fully exclude Crohn's disease (which has a more recognised association with coeliac disease) but the greater disease activity in the rectum compared with the sigmoid when assessed visually would support UC. There were no features to suggest microscopic colitis.

Duodenal histology revealed an increase in intraepithelial lymphocytes and inflammation of the lamina propria. There were some flattened villi, but some were preserved. These findings are in keeping with coeliac disease.

On discussion none of the Consultants felt that they would have thought to order TGA antibodies while investigating for IBD.

At the MDT meeting the team discussed the possibility of producing an initial advice sheet for surgeons to issue in such cases, but the lower GI nurse has recently returned from maternity leave so reports can be copied to her to enable her to follow up the patients using urgent clinic slots which are available quite quickly.

FB is currently awaiting full colonoscopy to assess the extent of her colitis and hopefully finally clarify the diagnosis, and to ascertain whether oral treatment will be indicated. However in view of the response to enemas this may turn out to be localised disease. She was advised by the gastroenterologist to reduce the enemas to alternate day use once her symptoms had resolved, however bleeding recurred on this regime so she is now using daily enemas again.

A DEXA scan ordered by her GP showed osteopenia. Her gynaecological history gives no suggestion of any previous oestrogen deficiency; she has had regular monthly periods until a slight reduction in frequency over the past 6 months. She has been given advice regarding weight bearing exercise and prescribed calcium & Vitamin D supplements no a long term basis. We will consider a repeat DEXA in a few years' time or at the menopause to plan for possible oestrogen replacement.

She was referred to hospital coeliac dietician service, but by the time she was seen had already joined Coeliac UK and started a gluten free diet with the help of her husband, who is an excellent cook. She described a resolution of her symptoms of bloating and the frequent passage of loose stools but identified some episodes of tiredness. The dietician identified potential sources of contamination in her diet and gave advice about avoiding these. She was informed of her entitlement to gluten free products on prescription, but prefers instead to adjust her diet to incorporate different foods.

Blood tests taken in the clinic revealed that the TGA antibodies had already returned to the normal range (2.3) but that her ferritin level was marginally reduced, so she has been prescribed iron in a once daily dose only, to avoid exacerbating her symptoms.

We are in the process of arranging the recommended vaccinations (she had Pneumococcus & influenza last autumn). She has been encouraged to speak to family members about arranging screening and thinks from their symptoms that at least one or two may turn out to have coeliac disease.

Permission has been given by FB to use her case.

INTRODUCTION

- *How do you get patients to take their medicine? The situation is particularly difficult when you have a medication that keeps people well but does have obvious and/or immediate problems if stopped. It can be very difficult to enthuse and encourage patients to continue their medication in IBD when they are feeling well. The age group affected is often the most difficult to encourage so Tillotts, one of our sponsors, has set up a website which may be of help in their regard. The accompanying text explains more.*

SECTION 6

"The Quest for Compliance - A New Patient Support Website in UC"

David Impey,Tillott's pharmaceuticals.

There's an adage in pharmaceutical marketing that the most expensive drug in the world is one the patients don't take. This is very true in Ulcerative Colitis.

The problem, to a great extent, is the patient demographic. The majority of UC sufferers are diagnosed in their teens or early twenties when, frankly, there are other more pressing distractions in life – career, college, relationships, social, etc. etc. Given that UC can be quiescent proportionately more than active, taking medication tends to fall in priority in comparison.

The long term consequences

Sunandra Kane, in one of her reviews of IBD, found that roughly half of patients are compliant to therapy (where compliance is defined as taking drug ‘more than 80% of the time’). This leaves half of the patients not being compliant thus exposing them to the increased risk of relapse, hospitalisation, surgery and possibly colorectal cancer.

Whilst the proportion of non-compliant patients who go on to have these serious sequelae is thankfully small, the resources – both financial and human – involved in their additional treatment is high. Any reduction therefore in these sequelae will be beneficial.

Active involvement

It is the experience of this writer that poor compliance tends to go hand-in-hand with poor knowledge of the condition. Therefore, one possible answer to the problem of poor compliance is better education. This has been the experience in a number of different therapeutic areas - including those with a dramatically different demographic – such as benign prostatic hyperplasia. Very simply, understanding on the part of the patient leads to the confidence to take a more active role in self-management.

The key was to determine what level of patient education in UC was out there. Initial trawls of the internet revealed that there was some information available via the Crohn’s and Colitis UK website although not specifically geared to the younger audience. The alternatives were either very academic medical websites or US sites such as WebMD which, again, tended to be either technical or possibly frightening.

The next step was to run focus groups with young patients. Patients were recruited through an IBD Nurse and two groups were run in London and Birmingham. In all, 15 patients aged between 18 and 28 participated. The results were revealing, not to say surprising.

What the patients said

The first thing to emerge from the groups was that this was the first time any of the patients had sat in a room with other patients to discuss their experiences. Quite quickly, the focus groups turned into encounter groups. They had all had similar diagnostic journeys with all patients – bar one – stating that the first diagnosis they had been given was piles.

The average time from first presentation to a GP to confirmed diagnosis by a GI specialist was 6 months (including the average 2 month wait for a secondary care appointment) with one or two getting the definitive diagnosis as quickly as 3 months and a similar number having to wait in excess of 9 months.

When asked about the quality of information available to them, the consensus was that there was very little provided to them by secondary care – although IBD nurses came in for great praise, but getting hold of them was tough. The information on the internet was, frankly, off-putting and several patients stated that they’d rather not know than dig any deeper.

What the patients wanted

On this point, the focus groups were very forthcoming. The key areas where it was felt that information was lacking – or at least not very reassuring – was about the disease itself, what the future held for them and more frankly, practical information on living with the disease. This included information on drinking, diet, socialising (what do they tell girl/boyfriends), coping with college / work / family.

UCandme.co.uk website

On the strength of the responses from the patient groups, it was decided to build a new patient website specifically targeted at the needs of the younger UC patients and use the website as a means of augmenting the launch of our brand of mesalazine. In this way, patients who were prescribed the brand could have access to the website (this is through the registration process in which patients are asked for the Batch Number and Expiry Date printed on the packaging – healthcare professional can gain access by entering a GMC or NMC number).

The site was deliberately un-healthcare like (see picture) in that the visitor would be presented with a street, the name of which was personalised as per their registration. Each building would offer new information as they progressed along the street. For example: the Bookshop would give information on UC, enable the visitor to print off letters to give to key people at college or work and see videos of fellow sufferers talking about their experiences; the gym, on the other hand, would give information on diet, coping with stress and keeping fit. And so on…

It was also decided to offer as many links to external sites including Crohn's and Colitis UK, to which patients registered with UCandme.co.uk can get 6 months' free membership.

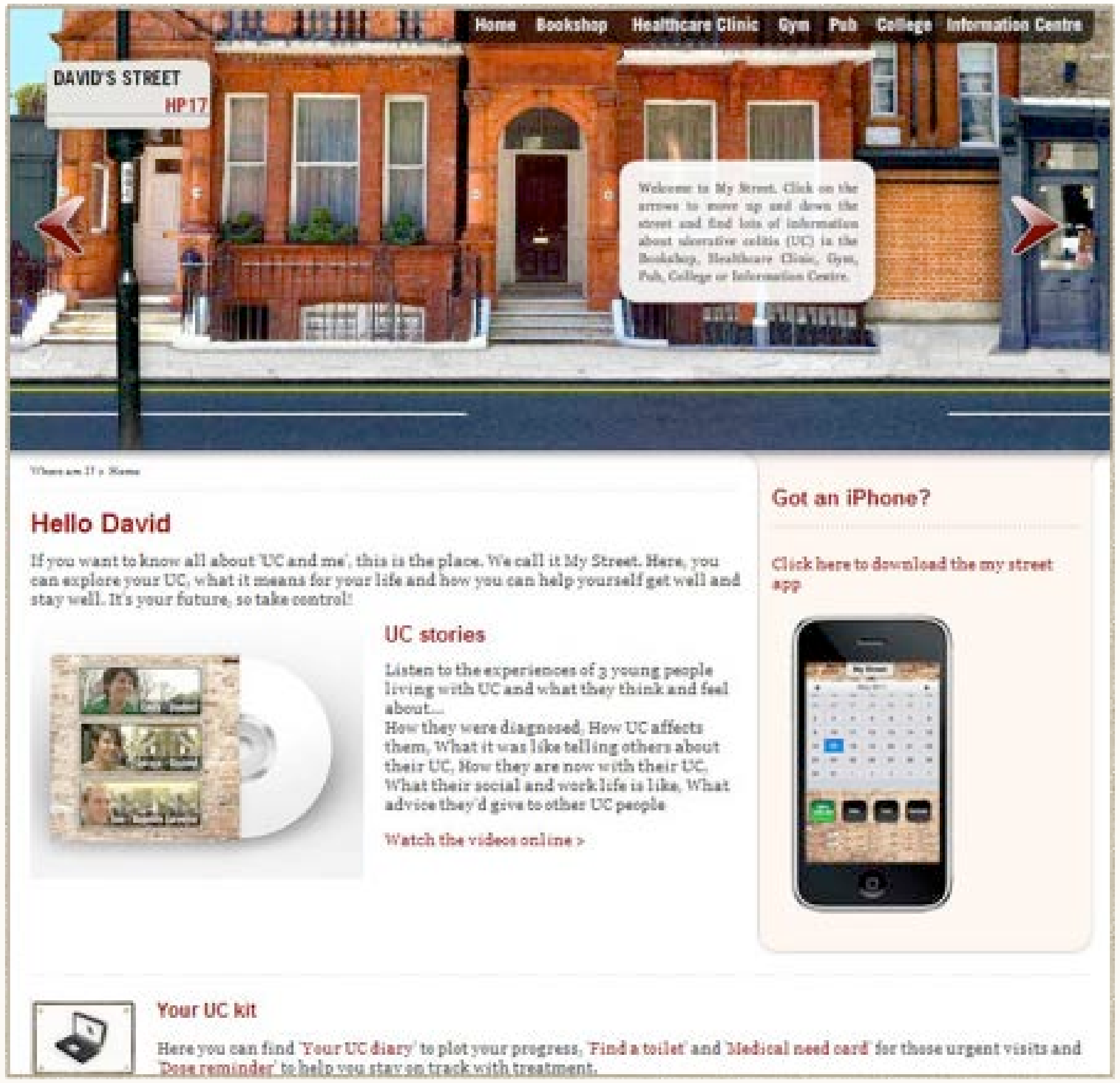
The material for the site was prepared by an advertising agency with significant input from a GI consultant, an IBD nurse and a dietician and the final output was reviewed by a panel of GI specialists. The site went live in June 2011 and has received a great deal of praise from healthcare specialists, patients and – even – the MHRA.

And finally…

It should be stated that the website was built as a means of providing product support ('after sales service' if you prefer) to Tillotts Pharma UK's brand of mesalazine Octasa.

That said, the website is completely non-promotional and strictly confidential. Tillotts do not have any access to the details of the visitors and will not use the site to make any approaches to them – this was a strict condition of the MHRA as access to the site is referred to on the Patient Information Leaflet.

Please visit the site. Tillotts, as sponsors of the PCSG, would be delighted to know your views.



Welcome to My Street. Click on the arrows to move up and down the street and find lots of information about ulcerative colitis (UC) in the Bookshop, Healthcare Clinic, Gym, Pub, College or Information Centre.

Where am I? > Home

Hello David

If you want to know all about 'UC and me', this is the place. We call it My Street. Here, you can explore your UC, what it means for your life and how you can help yourself get well and stay well. It's your future, so take control!

UC stories

Listen to the experiences of 3 young people living with UC and what they think and feel about...
How they were diagnosed, How UC affects them, What it was like telling others about their UC, How they are now with their UC, What their social and work life is like, What advice they'd give to other UC people

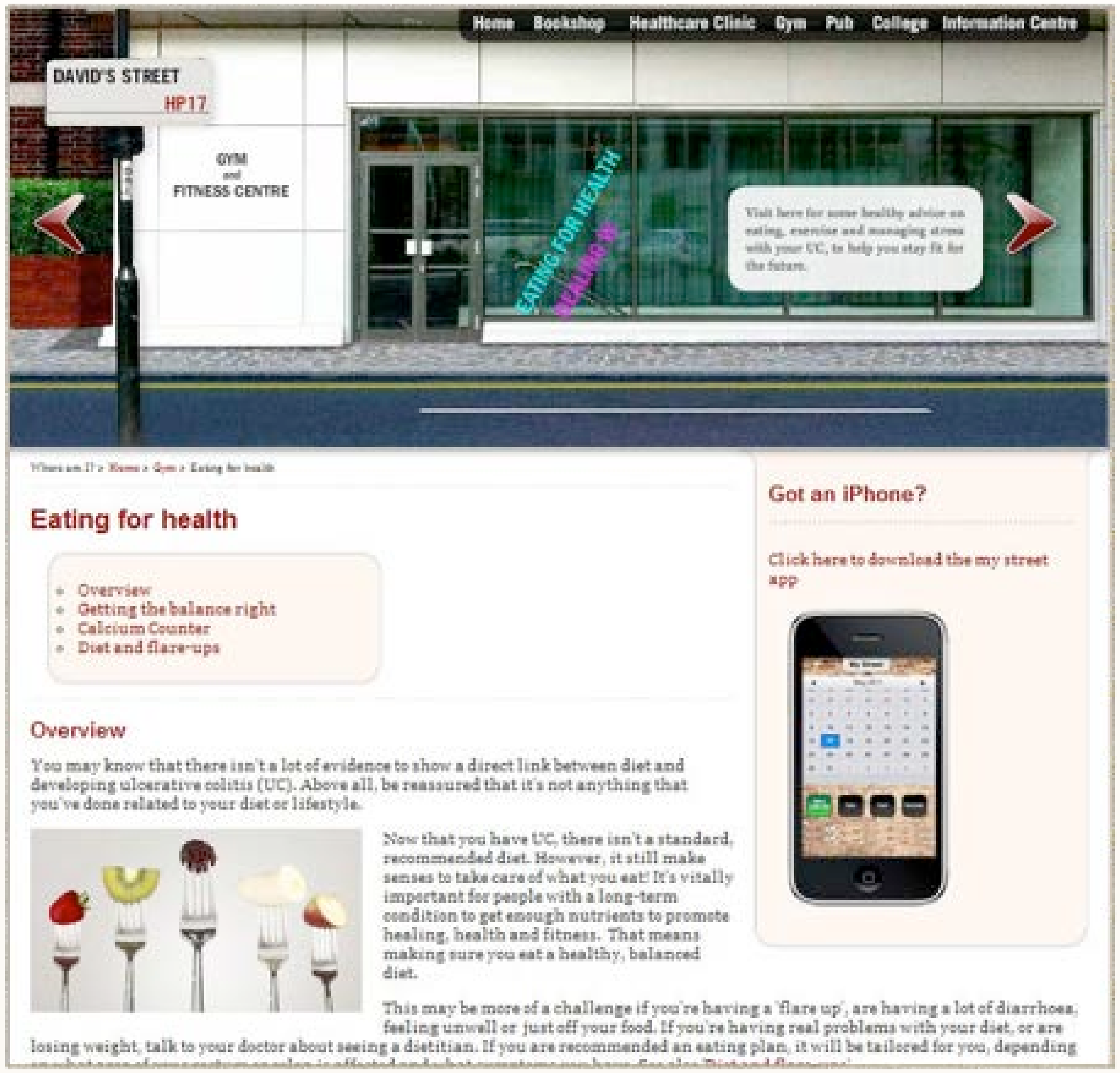
Watch the videos online >

Got an iPhone?

Click here to download the my street app

Your UC kit

Here you can find 'Your UC diary' to plot your progress, 'Find a toilet' and 'Medical need card' for those urgent visits and 'Dose reminder' to help you stay on track with treatment.



Visit here for some healthy advice on eating, exercise and managing stress with your UC, to help you stay fit for the future.

Where am I? > Home > Gym > Eating for health

Eating for health

- Overview
- Getting the balance right
- Calcium Counter
- Diet and flare-ups

Overview

You may know that there isn't a lot of evidence to show a direct link between diet and developing ulcerative colitis (UC). Above all, be reassured that it's not anything that you've done related to your diet or lifestyle.

Now that you have UC, there isn't a standard, recommended diet. However, it still make senses to take care of what you eat! It's vitally important for people with a long-term condition to get enough nutrients to promote healing, health and fitness. That means making sure you eat a healthy, balanced diet.

This may be more of a challenge if you're having a 'flare up', are having a lot of diarrhoea, feeling unwell or just off your food. If you're having real problems with your diet, or are losing weight, talk to your doctor about seeing a dietitian. If you are recommended an eating plan, it will be tailored for you, depending

Got an iPhone?

Click here to download the my street app

The society would like to acknowledge support from the following members of the Corporate Membership Scheme.

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If there are any subjects you would like covered in future issues, case histories you would like discussing or even articles you have penned yourself, send them to John O'Malley, Editor JPCSG at johnomalley@nhs.net.

Thanks.