Mimics of Inflammatory Bowel Disease

Dr Catriona McKenzie
Royal Prince Alfred Hospital, Camperdown
Mimics of IBD: Overview

- Infections
- Drugs
- Autoimmune
- Other
## Infections

### Infectious mimics of IBD

<table>
<thead>
<tr>
<th>IBD mimic</th>
<th>Cause(s)</th>
<th>Populations at risk</th>
<th>Diagnostic clues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial enteropathogens</td>
<td><em>Salmonella, Shigella, Campylobacter, Yersinia, E. coli, Aeromonas</em></td>
<td><em>Infections are often food- or water-borne</em></td>
<td><em>Positive cultures/PCR</em></td>
</tr>
<tr>
<td></td>
<td><em>M. tuberculosis, Mycobacterium avium-intercellulare complex</em></td>
<td><em>Tuberculous colitis occurs regardless of immune status</em></td>
<td><em>Positive cultures/PCR</em></td>
</tr>
<tr>
<td>Mycobacterial infections</td>
<td></td>
<td><em>Non-tuberculous colitis affects immunodeficient patients</em></td>
<td><em>Acid-fast stains have low sensitivity in paraffin-embedded tissue sections</em></td>
</tr>
<tr>
<td>Sexually transmitted proctocolitis</td>
<td><em>Syphilis (T. pallidum), lymphogranuloma venereum (LGV, C. trachomatis serotypes L1, L2, L3)</em></td>
<td><em>HIV-infected men who have sex with men</em></td>
<td><em>Positive serologic studies (syphilis)</em></td>
</tr>
<tr>
<td>Amoebiasis</td>
<td><em>Entamoeba histolytica</em></td>
<td><em>All ages affected</em></td>
<td><em>Positive culture/PCR (LGV)</em></td>
</tr>
<tr>
<td>Cord colitis</td>
<td><em>Bradyrhizobium enterica</em> (presumably)</td>
<td><em>Worldwide distribution</em></td>
<td><em>25–40 nm, PAS + trophozoites containing erythrocytes within adherent exudate</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Cord blood hematopoietic stem cell transplant recipients</em></td>
<td><em>Positive E. histolytica serology</em></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><em>Culture-negative, antibiotic-responsive diarrhoea</em></td>
</tr>
</tbody>
</table>
Case 1: NSW does it better

- 46M
- Diagnosed with ulcerative colitis in “the country” on biopsy
- Refractory to treatment with steroids and infliximab
- Transferred to RPAH where he underwent an emergency proctocolectomy
Previous biopsies
Entamoeba histolytica

- Parasitic infection, oro-faecal transmission
- 10% of world’s population is infected
- Industrialised countries – immigrants, returned travellers, MSM
- Symptoms vary from vague to fulminant colitis
- May form mass (ameboma)
- May disseminate (liver, CNS)
24 cases identified of intestinal amoebiasis with concomitant corticosteroid therapy

25% cases fatal

11 (46%) given steroids for misdiagnosed IBD

Organisms identified on histology on 47% cases from endoscopic biopsies
Learning points from a case of severe amoebic colitis

Christina Petridou¹, Adnan Al-Badri², Anjana Dua¹, Matthew Dryden¹, Kordo Saeed¹
¹Microbiology Department, Hampshire Hospitals NHS Foundation Trust, Royal Hampshire County Hospital, Winchester, United Kingdom;
²Pathology Department, Hampshire Hospitals NHS Foundation Trust, Royal Hampshire County Hospital, United Kingdom
Three subsequent cases

- 67 M
- Cardiac arrest, perforation with 4 quadrant peritonitis

- 75 M
- Perforation and peritonitis
58 M, Caecal “tumour”
Case 2: Wear a mask

- 61 year old man
- Right hemi colectomy
Further history/ subsequent course

- No history of IBD or recent overseas travel
- ZN (and other organisms stains) negative
- Mycobacterial PCR negative
- Culture of peritoneal nodules negative
- Treated presumptively for TB

Additional findings:

- AFB Culture (includes AFB Stain - Accession: 00000M0B20150139490)

  - Micro Reports
    - Specimen
      - Mycobacterium tuberculosis complex isolated
      - For further results and/or susceptibilities refer to report:
        - MB-15-139342
        - Specimen collected: 8/5/2015 at: 19.23
      - Preliminary Report - 29 May 2015 11:52 -
        - Mycobacterium tuberculosis complex (presumptive) isolated
        - Identification to follow
        - For further results and/or susceptibilities refer to report:
          - MB-15-139342
          - Specimen collected: 8/5/2015 at: 19.23
      - Microscopy - 11 May 2015 14:59 -
        - No acid fast bacilli seen
• 20 year period 841 patients identified with TB, 2.4% abdominal involvement, <1% ileo-caecal or small bowel involvement
Review article: the diagnosis and management of Crohn’s disease in populations with high-risk rates for tuberculosis

D. Epstein*, G. Watermeyer† & R. Kirsch‡ © 2007 The Authors, Aliment Pharmacol Ther 25, 1373–1388

Table 2. Prevalence of selected histological parameters in patients with intestinal tuberculosis (ITB) and Crohn’s disease (CD): A comparison of three similar studies.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>ITB (n = 20)</td>
<td>CD (n = 20)</td>
<td>ITB (n = 33)</td>
</tr>
<tr>
<td></td>
<td>ITB (n = 33)</td>
<td>CD (n = 31)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ITB (n = 18)</td>
<td>CD (n = 25)</td>
<td></td>
</tr>
<tr>
<td>Caseous necrosis</td>
<td>40</td>
<td>0</td>
<td>36</td>
</tr>
<tr>
<td>Confluent granulomas</td>
<td>60</td>
<td>0</td>
<td>42</td>
</tr>
<tr>
<td>≥5 granulomas/biopsy site</td>
<td>40</td>
<td>0</td>
<td>45</td>
</tr>
<tr>
<td>≥10 granulomas/biopsy site</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Large granulomas</td>
<td>Diameter &gt; 200 μm</td>
<td>90</td>
<td>51</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>61</td>
</tr>
<tr>
<td>Submucosal granulomas</td>
<td>45</td>
<td>5</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>61</td>
</tr>
<tr>
<td>Ulcers lined by bands of epithelioid histiocytes</td>
<td>45</td>
<td>5</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>61</td>
</tr>
<tr>
<td>Disproportionate submucosal inflammation</td>
<td>65</td>
<td>5</td>
<td>- 67</td>
</tr>
<tr>
<td>Architectural distortion distant to granulomatous inflammation</td>
<td>-</td>
<td>-</td>
<td>0  62</td>
</tr>
</tbody>
</table>

Values are given in percentages.
Is Crohn’s disease caused by a mycobacterium? Comparisons with leprosy, tuberculosis, and Johne’s disease

Robert J Greenstein

Detection of organisms with ZN and PCR in ITB

• Reports of AFB positivity on ZN in ITB variable 6-8% to 53.4%

• Reported sensitivity rate of PCR on FFPE tissue of endoscopic biopsies of ITB 22-75%

Epstein et al. Aliment Pharmacol Ther 2007
Dasgupta et al. J Lab Physicians 2009
Case 3: Sexually transmitted infectious proctitis/colitis

- Syphilis and LGV/CT most commonly reported
- Neisseria gonorrhea

Reminder of important clinical lesson

Lymphogranuloma venereum and HIV infection: misdiagnosed as Crohn’s disease

Sheel Patel, Phillip Hay

BMJ Case Reports 2010; doi:10.1136/bcr.02.2010.2771
Sexually Transmitted Infectious Colitis vs Inflammatory Bowel Disease

Distinguishing Features From a Case-Controlled Study

Christina A. Arnold, MD,1 Rachel Roth, MD,1 Razvan Arsenescu, MD,2 Alan Hargman, MD,3 Dora M. Lam-Himlin, MD,4 Berkeley N. Limketkai, MD,5 Elizabeth A. Montgomery, MD,6 and Lysandra Voltaggio, MD6

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**Venn Diagram**

- **STI Colitis**
  - Endoscopic impressions
  - Upper tract involvement
  - Terminal ileum involvement
  - "Skip lesions"
  - Erosions
  - Ulcerations
  - Aphthoid lesions
  - Increased mucosal chronic inflammation
  - Submucosal plasma cells, endothelial swelling, and perivascular plasma cells

- **IBD**
  - Prominent active chronic crypt centric damage
  - Prominent mucosal eosinophilia
  - Paneth cell metaplasia
  - Lymphoid aggregates
  - Granulomata
  - Fibrosis
  - Mucosal plasma cells, endothelial swelling, and perivascular plasma cells

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*Am J Clin Pathol 2015;144:771-781*
56M. Diarrhoea, weight loss. Anal ulcer
Diagnoses:  Early syphillis infection (with concurrent features of both primary & secondary syphillis)
Anorectal gonorrhoea infection
Fifty shades of chronic colitis: non-infectious imposters of inflammatory bowel disease

- Infections
- Drugs
- Autoimmune
- Other
Case 4 #quite severe

- 69 year old man
- Pan colitis
- ?Toxic megacolon on imaging
- Known metastatic melanoma on treatment with Ipilimumab
# Immune Checkpoint Inhibitor-Induced Colitis: Diagnosis and Management

Caroline Prieux-Klotz\textsuperscript{1} • Marie Dior\textsuperscript{1,2} • Diane Damotte\textsuperscript{3} • Johann Dreanic\textsuperscript{1,2} • Bertrand Brieau\textsuperscript{1,2} • Catherine Brezault\textsuperscript{1,2} • Vered Abitbol\textsuperscript{1} • Stanislas Chaussade\textsuperscript{1,2} • Romain Coriat\textsuperscript{1,2}

Targ Oncol (2017) 12:301–308

## Table 1
Rate of gastrointestinal side effects in anti-PD-1, anti-PD-L1, and anti-CTLA4 clinical trials

<table>
<thead>
<tr>
<th>Author and publication</th>
<th>Treatment</th>
<th>N</th>
<th>Gastrointestinal toxicities: all grade N(%)</th>
<th>Grade III–IV gastrointestinal toxicities N(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hodi et al. [5]</td>
<td>Ipilimumab 3 mg/kg every 3 weeks</td>
<td>131</td>
<td>Diarrhea: 36 (25.7%)</td>
<td>Diarrhea: 6 (4.6%) Colitis: 10 (7.6%)</td>
</tr>
<tr>
<td>Hodi et al. [5]</td>
<td>Ipilimumab 3 mg/kg + glycoprotein 100 mg every 3 weeks</td>
<td>380</td>
<td>Diarrhea: 115 (30.3%) Colitis: 20 (5.3%)</td>
<td>Diarrhea: 14 (3.7%) Colitis: 12 (3.1%)</td>
</tr>
<tr>
<td>Robert et al. [27]</td>
<td>Pembrolizumab 2 mg/kg every 2 weeks</td>
<td>89</td>
<td>0</td>
<td>Diarrhea: 1 (1.2%)</td>
</tr>
<tr>
<td>Robert et al. [27]</td>
<td>Pembrolizumab 10 mg/kg every 3 weeks</td>
<td>84</td>
<td>Diarrhea: 1 (1.2%)</td>
<td>Diarrhea: 1 (0.6%)</td>
</tr>
<tr>
<td>Garon et al. [9]</td>
<td>Pembrolizumab 2 mg/kg or 10 mg/kg every 2 or 3 weeks</td>
<td>495</td>
<td>Diarrhea: 40 (8.1%)</td>
<td>Diarrhea: 1 (0.3%) Colitis: 2 (0.7%)</td>
</tr>
<tr>
<td>Robert et al. [6]</td>
<td>Nivolumab 3 mg/kg every 2 weeks</td>
<td>206</td>
<td>Diarrhea: 39 (16%) Colitis: 2 (1%)</td>
<td>Diarrhea: 1 (0.3%) Colitis: 2 (0.7%)</td>
</tr>
<tr>
<td>Weber et al. [7]</td>
<td>Nivolumab 3 mg/kg every 2 weeks</td>
<td>268</td>
<td>Diarrhea: 30 (11.2%) Colitis: 3 (1.1%)</td>
<td></td>
</tr>
<tr>
<td>Rizvi et al. [10]</td>
<td>Nivolumab 3 mg/kg every 2 weeks</td>
<td>117</td>
<td>Diarrhea: 12 (10.5%)</td>
<td>Diarrhea: 12 (10.5%)</td>
</tr>
<tr>
<td>Rittmeyer et al. [28]</td>
<td>Atezolizumab 1200 mg every 3 weeks</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
• Neutrophilic infiltrates with cryptitis, crypt abscesses, +/-granulomas
• Lymphocytic infiltrate
• Mixed
• Increased apoptosis/GVHD like
• Small Bowel involvement
### Pembrolizumab associated colitis

#### Table 1: Patients without Ipilimumab Exposure

<table>
<thead>
<tr>
<th>No Ipilimumab Exposure</th>
<th>Nivolumab</th>
<th>Pembrolizumab</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of Patients</td>
<td>8</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Median Age</td>
<td>61.5</td>
<td>64</td>
<td>62</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>5 (62)</td>
<td>0 (0)</td>
<td>5 (42)</td>
</tr>
<tr>
<td>Female, n (%)</td>
<td>3 (38)</td>
<td>4 (100)</td>
<td>7 (58)</td>
</tr>
<tr>
<td>Median doses of drug</td>
<td>13.5</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>Median time to symptoms from drug initiation (months)</td>
<td>8.5</td>
<td>4</td>
<td>6.5</td>
</tr>
</tbody>
</table>

#### Imaging: n (%)

<table>
<thead>
<tr>
<th></th>
<th>Nivolumab</th>
<th>Pembrolizumab</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>4 (67)</td>
<td>1 (33)</td>
<td>5 (56)</td>
</tr>
<tr>
<td>Colitis</td>
<td>2 (33)</td>
<td>2 (67)</td>
<td>4 (44)</td>
</tr>
</tbody>
</table>

#### Endoscopy: n (%)

<table>
<thead>
<tr>
<th></th>
<th>Nivolumab</th>
<th>Pembrolizumab</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>6 (67)</td>
<td>1 (25)</td>
<td>7 (54)</td>
</tr>
<tr>
<td>Mild congestion, edema, inflammation</td>
<td>1 (11)</td>
<td>1 (25)</td>
<td>2 (15)</td>
</tr>
<tr>
<td>Moderate congestion, edema, inflammation</td>
<td>2 (22)</td>
<td>1 (25)</td>
<td>3 (23)</td>
</tr>
<tr>
<td>Severe congestion, edema, inflammation</td>
<td>0 (0)</td>
<td>1 (25)</td>
<td>1 (8)</td>
</tr>
</tbody>
</table>

#### Pathology: n (%)

<table>
<thead>
<tr>
<th></th>
<th>Nivolumab</th>
<th>Pembrolizumab</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>4 (44)</td>
<td>1 (25)</td>
<td>5 (38)</td>
</tr>
<tr>
<td>Lymphocytic Colitis</td>
<td>1 (11)</td>
<td>0 (0)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Collagenous Colitis</td>
<td>0 (0)</td>
<td>1 (25)</td>
<td>1 (8)</td>
</tr>
<tr>
<td>Active colitis</td>
<td>4 (44)</td>
<td>2 (50)</td>
<td>6 (46)</td>
</tr>
<tr>
<td>Other</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
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</tbody>
</table>

#### Treatment: n (%)

<p>| | | | |</p>
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</thead>
<tbody>
<tr>
<td>Steroids</td>
<td>7 (87.5)</td>
<td>4 (100)</td>
<td>11 (92)</td>
</tr>
<tr>
<td>Infliximab</td>
<td>1 (12.5)</td>
<td>2 (50)</td>
<td>3 (25)</td>
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</table>

#### Outcome: n (%)

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<tr>
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</thead>
<tbody>
<tr>
<td>Symptom resolution</td>
<td>8 (100)</td>
<td>4 (100)</td>
<td>12 (100)</td>
</tr>
<tr>
<td>Drug discontinued</td>
<td>5 (62.5)</td>
<td>3 (75)</td>
<td>8 (67)</td>
</tr>
<tr>
<td>Treatment-related death</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

Kim et al, AGA 2017 (abstract)
Pembrolizumab associated colitis
Baseline gut microbiota predicts clinical response and colitis in metastatic melanoma patients treated with ipilimumab

N. Chaput\textsuperscript{1,2\dagger}, P. Lepage\textsuperscript{3\dagger}, C. Coutzac\textsuperscript{1,4}, E. Soularue\textsuperscript{1,4,5}, K. Le Roux\textsuperscript{3}, C. Monot\textsuperscript{3}, L. Boselli\textsuperscript{1}, E. Routier\textsuperscript{6}, L. Cassard\textsuperscript{1}, M. Collins\textsuperscript{4,5}, T. Vaysse\textsuperscript{4,5}, L. Marthe\textsuperscript{4,5}, A. Eggermont\textsuperscript{6,7}, V. Asvatourian\textsuperscript{8,9}, E. Lanoy\textsuperscript{8,9}, C. Mateus\textsuperscript{4}, C. Robert\textsuperscript{4,6\dagger} & F. Carbonnel\textsuperscript{4,5\dagger}
Rapid complete response of metastatic melanoma in a patient undergoing ipilimumab immunotherapy in the setting of active ulcerative colitis

A Doran Bostwick\textsuperscript{1}, April K Salama\textsuperscript{2} and Brent A Hanks\textsuperscript{2*}
Mimics of IBD

- Infections
- Drugs
- Autoimmune
- Other
**Case 5. What’s missing?**

**CLINICAL DETAILS**

- 74F
Colonic biopsies
Duodenal biopsies
Autoimmune enteropathy

- Rare (<1/100000 infants)
- First described in 1982 in children, now well recognised in adults
  - Syndromic
    - IPEX (Immune dysregulation, Polyendocrinopathy, Enteropathy and X linked) - loss of function mutations in FoxP3
    - APECED syndrome (Autoimmune Polyendocrinopathy Candidiasis Ectodermal Dystrophy Syndrome)
  - Non syndromic

Further history

• 74 F presented to Dubbo March 2014 with 3 month history of severe diarrhoea
• Transferred to RPA - responded to steroids, TPN and Sulphasalazine, returned to Dubbo
• Diarrhoea returned with fevers, complicated by documented CMV, underwent emergency colectomy and end ileostomy
• Steroids reduced again but high stoma output (2-3L) day – re- biopsied
Progress

• Diagnosis of autoimmune enteropathy
• Recommenced high dose steroids and azathioprine
• Plasma sent to Children’s Hospital of Philadelphia for anti-enterocyte antibody testing (negative)
• Protracted diarrhoea
• Small intestinal villous atrophy
• Lack of response to dietary therapy
• Evidence of autoimmunity (circulating autoantibodies to gut epithelium/associated autoimmune diseases)

Masia et al, AJSP 2014
Talia L. Fuchs¹, Jeffrey L. Engelman², Mark Tschuchnigg¹

¹ Australian Clinical Labs, Bella Vista, Sydney Australia
² Gastroenterologist, St George Private Hospital, Sydney Australia

Figure 7: Anti-enterocyte IgG autoantibodies immunofluorescence 1:16 dilution (20x magnification). Image courtesy of A/Prof Lynette Moore, SA Pathology, Adelaide (2016).
Case 6. What’s missing #2?

<table>
<thead>
<tr>
<th>Male</th>
<th>31 years</th>
</tr>
</thead>
</table>

**CLINICAL DETAILS**

Diagnosis

CVID

.......and recent c.jejuni infection
Gastrointestinal Tract Pathology in Patients With Common Variable Immunodeficiency (CVID)

A Clinicopathologic Study and Review

Jason A. Daniels, MD,* Howard M. Lederman, MD, PhD,† Anirban Maitra, MBBS,* and Elizabeth A. Montgomery, MD*

TABLE 2. Percentage of Patients Showing Histologic Abnormality

<table>
<thead>
<tr>
<th>Histologic Findings</th>
<th>Esophagus</th>
<th>Stomach</th>
<th>Small Intestine</th>
<th>Colon</th>
<th>Appendix</th>
<th>Gallbladder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased plasma cells</td>
<td>N/A</td>
<td>12/18 (67%)</td>
<td>13/19 (68%)</td>
<td>10/16 (63%)</td>
<td>1/1 (100%)</td>
<td>1/2 (50%)</td>
</tr>
<tr>
<td>Lymphoid aggregates</td>
<td>N/A</td>
<td>11/18 (61%)</td>
<td>9/19 (47%)</td>
<td>13/16 (81%)</td>
<td>0/1 (0%)</td>
<td>0/2 (0%)</td>
</tr>
<tr>
<td>Increased apoptosis</td>
<td>1/10 (10%)</td>
<td>6/18 (33%)</td>
<td>4/19 (21%)</td>
<td>8/16 (50%)</td>
<td>1/1 (100%)</td>
<td>0/2 (0%)</td>
</tr>
<tr>
<td>Intraepithelial lymphocytosis</td>
<td>5/10 (50%)</td>
<td>4/18 (22%)</td>
<td>12/19 (63%)</td>
<td>6/16 (38%)</td>
<td>0/1 (0%)</td>
<td>0/2 (0%)</td>
</tr>
<tr>
<td>Villous blunting*</td>
<td>N/A</td>
<td>N/A</td>
<td>10/12 (83%)</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Collagenous pattern*</td>
<td>N/A</td>
<td>0/4 (0%)</td>
<td>0/12 (0%)‡</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Granulomas</td>
<td>0/10 (0%)</td>
<td>1/18 (6%)</td>
<td>2/19 (11%)</td>
<td>3/16 (19%)</td>
<td>0/1 (0%)</td>
<td>0/2 (0%)</td>
</tr>
<tr>
<td>Intraepithelial neutrophils</td>
<td>4/10 (40%)</td>
<td>8/18 (44%)</td>
<td>6/19 (32%)</td>
<td>14/16 (88%)</td>
<td>0/1 (0%)</td>
<td>1/2 (50%)</td>
</tr>
<tr>
<td>Crypt Distortion†</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Infections†</td>
<td>4/4 (100%)</td>
<td>2/8 (25%) CMV;</td>
<td>1/19 (5%)</td>
<td>1/14 (7%) CMV</td>
<td>0/0 (0%)‡</td>
<td>0/1 (0%)</td>
</tr>
<tr>
<td><strong>Cancer</strong></td>
<td>0/10 (0%)</td>
<td>1/18 (6%) AdenoCA</td>
<td>0/19 (0%)</td>
<td>0/16 (0%)</td>
<td>0/1 (0%)</td>
<td>0/2 (0%)</td>
</tr>
</tbody>
</table>

*When accompanied by intraepithelial lymphocytosis.
†When accompanied by intraepithelial neutrophils.
‡A single case of small intestinal mucosa with a collagenous pattern occurred in a patient without an increase in intraepithelial lymphocytosis.
§A single case of Cryptosporidium was identified in an appendix without neutrophilic infiltrate.

Crypt indicates Cryptosporidium; HP, H. pylori.
Mimics of IBD

- Infections
- Drugs
- Autoimmune
- Other

Top etiologic considerations of the chronic colitis pattern:

- Diverticular disease
- Diversion colitis
- Therapeutics
  - NSAIDs
  - Resins
  - Ipilimumab
- Vascular injury
  - Ischaemia
  - Radiation
  - Vasculitis
- Autoimmune
  - Sarcoid
  - Common variable immunodeficiency
  - Chronic granulomatous disease
  - Vasculitis
- Infections
  - Stool pathogens
  - Cord colitis syndrome
  - Syphilitic and lymphogranuloma venereum colitis
  - Others
Case 7: Diversion colitis

- 33 year old man. History of ulcerative colitis.
- Completion proctectomy
## Diversion colitis vs UC

<table>
<thead>
<tr>
<th>Feature</th>
<th>IBD of Pouch</th>
<th>Diversion Colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>IBD mucosal changes</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Crypt atrophy</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Lymphoid hyperplasia</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Diffuse disease</td>
<td>+/-</td>
<td>++</td>
</tr>
<tr>
<td>Previous IBD rectum</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Symptoms decreased on exclusion</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Symptoms decrease on hookup</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Ulcers</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Focal mild patchy cryptitis</td>
<td>+</td>
<td>++</td>
</tr>
</tbody>
</table>

- Absent; +, present.

Odze 2014
Case 8

- 68F
- Stage 3 breast cancer
- Recent colitis diarrhoea
- Colonoscopy + Bx -? diverticular associated colitis.
- Just affects sigmoid colon.
- Diverticular colitis?
12 months later........
“SCAD”
M>F, typically elderly
UC and Crohn’s like changes described
Usually managed conservatively
Reported to precede UC and Crohns
CLINICAL DETAILS
Unusual cystic foreign bodies through (R) colon. ?Parasite.
Commentary

Right lower quadrant pain in a farmer brings to mind many occupational diseases, among which certainly are parasites. Blastocystis, long considered a nonpathogen, is arguably now being appreciated as capable of causing GI distress, although it has no egg stage in its development and so could not account for the egglike forms seen at colonoscopy in this patient. In fact, these forms are too large for any of the parasitic eggs we see in daily practice throughout the world; perhaps if one were a gastroenterologist in the pre-Cambrian era, with the larger species seen then, well...who knows. These egglike forms were admirably identified by the authors as quinoa seeds. Quinoa, a species of goosefoot (Chenopodium), is thought to have been domesticated by the Incas in the Peruvian Andes 3000 to 4000 years ago; they held the crop to be sacred and referred to it as the “mother of all grains.” With the arrogance of conquerors, the conquistadors forbade quinoa cultivation and forced the Incas to grow wheat. Although quinoa is usually prized for its seeds, the green leaves of the quinoa growth also are edible, although not widely available. Chenopods are considered a pseudocereal rather than a true cereal because they are not a member of the true grass family; they are closely related to beetroot and spinach. Today, quinoa is very popular because it is gluten free and a very good source of protein and essential amino acids, magnesium, iron, and calcium. Who among us does not know a vegan, a lactose-intolerant person, or someone who is experimenting with a gluten-free diet and who would not benefit from this superfood? I am curious why the quinoa seeds were still present a week or 2 after ingestion. Was the patient’s memory about the time of its ingestion flawed? Does quinoa have an as-yet-undefined effect to decelerate intestinal motility? Isn’t it marvelous how observation of the unusual leads to speculation, which in turn promotes inquiry and research, that then advances our knowledge? One can learn so much about so many things by practicing medicine...and just paying attention.

Lawrence J. Brandt, MD
Associate Editor for Focal Points
Acknowledgments

- AGPS committee
- P Mckenzie
- J Shin
- T Dodds
- R Gupta
- E. Robbins
- L. Anderson
- J.Kench
- B. Elston
- T. Fuchs
- K. Seoane-Velilla
- C.Byrne
- C.Corte