Sarajevo November 2016

M15
1 year history of cervical lymphadenopathy
Recent onset of swollen lips
Cobblestone buccal mucosa
Oral mucosa biopsy
Orofacial granulomatosis

Histological features
- Intact epithelium
- Acanthosis
- Submucosal chronic inflammation
- Multiple non-caseating epithelioid granulomas
- Langhans-type giant cells
Orofacial granulomatosis

- Persistent/recurrent labial enlargement
- Oral ulcers
- Mucosal swelling (cobblestone)
- Also, mucosal tears, gingival enlargement, tongue fissures, facial nerve palsy, facial swelling/erythema, cervical lymphadenopathy
Orofacial granulomatosis

- Distinct entity
- Initial presentation of Crohn’s disease
- Incidence of IBD/CD relatively stable in Western world but increasing in Southern Europe, Asia and developing world
- Possible associations with atopy, food intolerance, hypersensitivity to dental materials
Orofacial granulomatosis

Differential Diagnosis
• Crohn’s disease
• Sarcoidosis
• Allergic angio-oedema
• Miescher’s cheilitis (Schuermann’s granulomatous cheilitis)
• Melkersson-Rosenthal syndrome
• Cheilitis glandularis
• Tuberculosis
Orofacial granulomatosis

Differential Diagnosis

• Crohn’s disease Gut involvement, HLA types
• Sarcoidosis Pulmonary involvement
• Allergic angio-oedema Clinical, atopy
• Miescher’s cheilitis (Schuermann’s granulomatous cheilitis) Isolated oral granulomas
• Melkersson-Rosenthal syndrome + LMN Facial nerve palsy
• Cheilitis glandularis Involvement of minor salivary glands
• Tuberculosis Immunosuppressed individuals
Orofacial granulomatosis

Reference

• Leao et al. Orofacial granulomatosis. Aliment Pharmacol Ther 2004; 20: 1019-1027
M76

History of dysphagia and gastro-oesophageal reflux disease

Stricture noted at 23 cm

Biopsy taken
Reflux oesophagitis

Reflux of gastric or duodenal contents into the oesophagus

- Associated with obesity, pregnancy, hiatus hernia, recurrent vomiting, large meals, alcohol, scleroderma
- Symptoms include heartburn, chest pain, acid taste, coughing and shortness of breath.
Reflux oesophagitis

Endoscopic findings

- Erosions
- Ulceration
- Exudates
- Stricture formation
- 50-60% of symptomatic patients with objective evidence of reflux disease have normal mucosa or at worst, hyperaemia
- Histologically inflamed mucosa may appear normal endoscopically
- Biopsy recommended in symptomatic patients to confirm inflammation and to exclude infections, Barrett’s mucosa and neoplasia
- Jumbo forceps biopsies are better than pinch forceps biopsies
- Reflux changes best seen in distal 8cm and above 2.5cm

From www.endoskopischer-atlas.de
Reflux oesophagitis

Pathological features
• Squamous hyperplasia
• Basal zone greater than 15% of epithelium
• Subepithelial papillae greater than $\frac{2}{3}$rd
• Correlates with pH and acid production
• Dilatation of capillaries
• Also ballooning degeneration, multinucleation
Reflux oesophagitis

Pathological features: inflammatory cells

• Neutrophils: should not normally be present but are an insensitive means of detecting reflux disease
• Lymphocytes: normally present, mainly T cells, and do not correspond well with reflux
• Eosinophils: good marker in paediatric practice, as not normally present, but may be seen in adults
Reflux oesophagitis

Differential diagnosis

- Infectious causes: Candida, CMV, HSV
- Corrosive oesophagitis (pills, bleach, acid)
- Iatrogenic oesophagitis (chemo/radiotherapy)
- Squamous dysplasia/neoplasia
- Systemic diseases (collagen-vascular disease, Stevens-Johnson syndrome, bullous diseases, lichen planus)
- GVHD
- Crohn’s disease
- Amyloidosis
- Trauma
Sarajevo November 2016

M60
History of dysphagia
Diffusely inflamed lower oesophagus
Random biopsies taken
Eosinophilic oesophagitis

Histological features
• Eosinophilic infiltrate
• Basal cell hyperplasia
Eosinophilic oesophagitis

Epidemiology

• Affects children and adults
• More common in males
• M:F Paediatric 2:1; Adults 7:3
• Middle aged males
Eosinophilic oesophagitis

Clinical: children
• Vague upper GI symptoms (younger)
• Dysphagia and food impaction
• History of atopy: asthma, rhinitis, dermatitis, eczema
Eosinophilic oesophagitis

Clinical: adults
• Dysphagia 93%
• Food impaction 62%
• Heartburn 24%
• Also non-cardiac chest pain, odynophagia, vomiting
Persistent dyspepsia/dysphagia on maximal acid suppression
Eosinophilic oesophagitis

Endoscopic

• Feline oesophagus (corrugated/ringed)
• Also furrows, strictures, exudates, narrowing, oedema, fragility
• May have eosinophilic gastritis or enteritis

From Furth et al AJR 1995 164: 900
Endoscopy (upper) of eosinophilic oesophagitis showing oesophageal ridging and furrows.

Maurice B Loughrey, and Brian T Johnston Frontline Gastroenterol 2014;5:88-95
Eosinophilic oesophagitis

Pathology
• Intense eosinophilic infiltration
• Single: > 20 eosinophils/hpf
• Multiple: > 15 eosinophils/hpf
• In GORD, usually < 7 eos/hpf
• Gradient to surface
• Microabscesses (>4 cell clusters)
• Degenration, slough
Eosinophilic oesophagitis

Pathology

• Also, ↑ T lymphocytes, mast cells, Langerhans cells
• Lamina propria fibrosis
• ↑ eosinophils in LP
• Other features: elongation of LP papillae, basal hyperplasia, acanthosis, oedema
Eosinophilic oesophagitis

Differential diagnosis
• GORD (distal)
• Parasitic, fungal infections
• Inflammatory bowel disease
• Hypereosinophilia syndrome
• Collagen vascular diseases
• Drug toxicity

Need for clinical information
Eosinophilic oesophagitis

Diagnosis

• Combination of clinical presentation, endoscopic features and histological appearances

• Atopic schoolboy or middle aged male with a history of dysphagia/food impaction despite maximal PPI therapy, furrowed oesophagus on endoscopy and eosinophils on histology
Eosinophilic oesophagitis

Reference
Chang F, Anderson S. Clinical and pathological features of eosinophilic oesophagitis: a review. Pathology 2008; 40: 3-8
Sarajevo November 2016

M41
Bone marrow transplant for leukaemia
History of dysphagia
Oesophageal biopsy for ? graft-versus-host disease
CMV Oesophagitis

- CMV part of herpes group (also HSV 1 and 2, EBV, VZV)
- Nuclear replication
- Latent infection
- Large intranuclear inclusions
- Smaller cytoplasmic inclusions
CMV Oesophagitis

- Worldwide 50-80% seropositive
- Preschool and young adults
- Immunosuppression, especially HIV
- Also, retinitis, adrenalitis, pneumonia, colitis
- CMV 3rd commonest HIV infection (PCP, candida)
- HAART reduced HIV complications
- Now, more commonly seen with other immunosuppression
CMV Oesophagitis

- Transplant (renal, solid organ, bone marrow)
- Other debilitating diseases
- CMV problems 5-7 months after solid organ tx
- CMV 2-3 months after BMT
CMV Oesophagitis

3 patterns of acquisition
• Primary: contact with seropositive individual in 60%, few symptoms
• Reactivation: latent infection in 10-20% of cases, associated with ↓ immunity
• Superinfection: contact with another seropositive individual in 20-40%

CMV persists indefinitely, controlled by cellular/humoral immunity, infection related to T cell activity
CMV Oesophagitis

Histology

• Variable
• Acute inflammation
• Chronic inflammation
• Ulceration
• Features of cytomegaly (large, ovoid, pleomorphic cells with basophilic inclusions)
CMV Oesophagitis

Clinical presentation
• Dysphagia
• Odynophagia
• Retrosternal pain
• Nausea and vomiting
• Abdominal pain
• Also, fever, diarrhoea, weight loss
CMV Oesophagitis

Differential diagnosis
• Achalasia
• Barrett’s oesophagus
• Candidiasis
• Cryptococcosis
• Carcinoma
• GORD
• HSV
• Histoplasmosis
• Tuberculosis
M79
History of anaemia and melaena
Raised lesions noted in lower oesophagus
Biopsies taken
HSV oesophagitis

Clinically DD
• GORD
• Infections
• Radiation rx
• Systemic diseases
• Trauma
• Immunosuppression (neutropenia, ↓ T cell function, steroids, radiation)
• Background disease: AIDS/HIV, DM, ↑ age
HSV oesophagitis

Clinically
• Dysphagia
• Odynophagia
• Heartburn
• Nausea and vomiting
• Fever
• Abdominal pain
• Epigastric pain
HSV oesophagitis

Differential diagnoses (infectious)
- Candidiasis
- Fungal infection
- HSV
- Herpes (CMV, VZV, EBV)
- HIV (MAI)
- HPV
- Bacterial
- Parasitic
HSV oesophagitis

Differential diagnoses (systemic-1)

• Epidermolysis bullosa
• Pemphigus vulgaris
• Bullous pemphigoid
• Drug response (EM, SJS, TEN)
• Lichen planus
• Acanthosis nigricans
• Leukoplakia
HSV oesophagitis

Differential diagnoses (systemic-2)
- Eosinophilic oesophagitis
- Behcet’s disease
- GVHD
- IBD (CD)
- Sarcoidosis
- Chronic granulomatous disease
- Drugs
- Rorx
HSV oesophagitis

Clinical

- Candida, CMV, VZV, EBV, HIV, TB, Drugs, Behcet’s
- GVHD: desquamation, webs, rings, strictures, apoptosis
- Rx: antivirals
HSV oesophagitis

DD: infections

• Rounded 1-3mm vesicles
• Distal oesophagus
• Raised ulcers
• Changes in epithelial cells
• MNGC, ballooning, ground glass, Cowdry A cells
History of dysphagia
Nodule noted at oesophago-gastric junction
Biopsies taken
Heterotopic pancreas in oesophagus

Finding of pancreatic parenchyma without anatomical or vascular connections to the native pancreas

Unusual condition in oesophagus: 0.2-0.5% of surgical resections; 0.6-13.6% of autopsies

Usually solitary

Intramural location, surface umbilication
Heterotopic pancreas in oesophagus

Pancreatic heterotopia relatively frequent elsewhere in GIT

- Stomach 25-93%
- Duodenum 28-36%
- Jejunum 15-47%
- Meckel’s diverticulum 5-6%
- Ileum 3%
- Reports in liver, gall bladder, mesentery, colon
Heterotopic pancreas in oesophagus

Pathology

• Finding of acini, islets of Langerhans and ducts
• Not just pancreatic acinar metaplasia

Most common in distal third/GOJ
Roughly male:female equality
May be diagnosed on FNAC
Heterotopic pancreas in oesophagus

Clinical

• GI bleeding
• Dysphagia, dyspepsia
• Boerhaave’s syndrome
• Mass lesion
• May be associated with congenital lesions: duplication cysts, diverticula, atresia and tracheo-oesophageal fistula
Heterotopic pancreas in oesophagus

Complications
• Pancreatitis
• Pseudocysts
• Rare cancers: adenocarcinoma, solid/papillary tumours, islet cell adenomas, anaplastic carcinoma
• Reported in stomach, jejunum, colon but not in oesophagus
Heterotopic pancreas in oesophagus

References


• Rodriguez et al. FNAC findings from a case of pancreatic heterotopia at the GEJ. Diagn Cytopathol 2004; 31: 175-179

M85
History of gastro-oesophageal reflux and recent dysphagia
Endoscopy showed abnormal ‘fleshy’ mucosa at 28cm
Biopsies taken
Sarajevo November 2016

M60
Previous history of Barrett’s mucosa
Follow up endoscopy showed polypoid mucosa at 34cm
Biopsies taken
Dysplasia in Barrett’s mucosa

Dysplasia is strictly synonymous with intraepithelial neoplasia

Diagnosis requires both cytological and architectural changes
Dysplasia in Barrett’s mucosa

Cytological changes
• nuclear alterations (size, shape, nucleoli)
• nuclear-cytoplasmic change
• hyperchromatism
• increased numbers of abnormal mitoses
Dysplasia in Barrett’s mucosa

Architectural changes

• glandular distortion and crowding
• intraluminal glandular papillary extensions
• villiform mucosal surface
Dysplasia in Barrett’s mucosa

Classification of dysplasia

• indefinite category
• increasing severity
• 3 (mild, moderate, severe) OR
• 2 (low or high) grades PREFERRED
Dysplasia in Barrett’s mucosa

Indefinite for dysplasia
• Epithelial atypia not diagnostic of dysplasia
• Associated with severe inflammation and ulceration
• Incomplete maturation
• Cytological features more marked than architectural changes
• Beware biopsy and processing artefacts
Dysplasia in Barrett’s mucosa

Indefinite for dysplasia

• Suggest repeat biopsies after treatment
• Discuss case with clinicians
• Need for follow up
Dysplasia in Barrett’s mucosa

Low grade dysplasia

• Some maturation from base to surface
• More cytological than architectural change
• Increased mitoses but not reaching surface
Dysplasia in Barrett’s mucosa

Low grade dysplasia

- Rate of progression to adenocarcinoma is uncertain but not negligible
- Absolute need for follow up
- Confirm diagnosis with another GI pathologist
- Regular endoscopy
- May be an indication for oesophagectomy or EMR or ablation therapy
- Discussion at MDTM
Dysplasia in Barrett’s mucosa

High grade dysplasia

- Villiform surface appearance
- Cytological changes including full thickness involvement
- Abnormal mitoses; prominent nucleoli; high N/C ratio; cellular pleomorphism
- Architectural changes: cribriform outline, back-to-back glands
Dysplasia in Barrett’s mucosa

High grade dysplasia

• Confirm diagnosis with another GI pathologist
• Discuss at MDT
• Management as for adenocarcinoma: decision on oesophagectomy dependent on staging and co-morbidities
• If unsuitable for definitive treatment, local treatment: EMR, laser therapy, stenting, radiotherapy
Iron deficiency anaemia. D2 biopsy

From endoscopy report: the whole upper GI tract was normal. Known coeliac disease; recent onset IDA whilst on gluten free diet. Distal duodenal biopsies taken to evaluate response to GFD.
Diagnosis

Coeliac disease showing partial response to GFD

Typical pattern of immunohistochemistry for T lymphocytes (CD3+, CD8+, CD4-)

No features of refractory disease
Villous atrophy in adults

Pathologic findings characteristic but not diagnostic
- Coeliac Disease
- Tropical Sprue
- Adult-onset autoimmune enteropathy
- Hypogammaglobulinemia
- Idiopathic
- AIDS enteropathy

Pathologic findings are non-specific
- Small-bowel bacterial overgrowth
- Infectious enteritis
- Parasitic infestation
- Severe malnutrition
- Small-bowel ischaemia

Pathologic findings could be diagnostic
- Eosinophilic gastroenteritis
- Whipple’s disease
- Abetalipoproteinaemia
- Intestinal lymphoma
- Collagenous sprue
- Tuberculosis
- Giardiasis
- Crohn’s disease
Can villous atrophy be diagnosed endoscopically or macroscopically?
MALABSORPTION-CAUSES

- Coeliac disease: extensive mucosal disease related to sensitivity to gluten
Serology for coeliac disease

• screen by TTG (check IgA levels)
• if positive, do EMA
• if positive, proceed to duodenal biopsy
• often duodenal biopsy is initial step (or not made aware of the results of serology)
• duodenal biopsy is the gold standard and things may go awry if CD is not so confirmed
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<th>Specificity</th>
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<td>90-95%</td>
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<td>IgA Good screen for CD</td>
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<td>anti-endomysial antibody (EMA)</td>
<td>90-95%</td>
<td>95%</td>
<td>IgA but really a crude TTG – needs very high titre</td>
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<td>anti-gliadin antibody</td>
<td>90% if IgA</td>
<td>80%</td>
<td>Depends on whether IgG or IgA measured</td>
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Upper Jejunal Mucosal Immunopathology

Pre-Infiltrative

Infiltrative

Infiltrative Hyperplastic

Flat Destructive

Atrophic Hypoplastic
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<th>IEL / 100 enterocytes - duodenum</th>
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<td>3c</td>
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<td>&gt;30</td>
<td>Increased</td>
<td>Complete atrophy</td>
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</table>

**Modified Marsh Classification of histologic findings in coeliac disease (Oberhuber)**

IEL/100 enterocytes, intraepithelial lymphocytes per 100 enterocytes

Type 0: Normal; coeliac disease highly unlikely.
Type 1: Seen in patients on gluten free diet (suggesting minimal amounts of gluten or gliadin are being ingested); patients with dermatitis herpetiformis; family members of coeliac disease patients, not specific, may be seen in infections.
Type 2: Very rare, seen occasionally in dermatitis herpetiformis.
Type 3: Spectrum of changes seen in symptomatic coeliac disease.
Quantitation of IELs in SI biopsies

- Traditionally <40 IELs/100 ECs (jejunal Crosby capsule biopsies)
- Re-evaluation in Western populations for endoscopic distal duodenal (D2) biopsies: normal <25 IELs/100 ECs
  

- Patchy villous atrophy may cause difficulties
- Suggestion to use duodenal bulb (D1) biopsies: recommendation is to take 4 x D2 + 1 x D1 at 9-12 o’clock

Kurien et al GI Endoscopy 2012; 75: 1190-6
The clinical significance of duodenal lymphocytosis with normal villous architecture

Increased intraepithelial lymphocytes with normal villous architecture (Marsh I lesion) seen in 3% of duodenal biopsies.

DDx: coeliac disease, bacterial overgrowth, NSAID damage, reaction to H. pylori infection, tropical sprue, CIBD

Marsh I lesions are a nonspecific finding associated with a number of disease conditions

Historically, 9-40% represent coeliac disease

Current data do not suggest histologic features to differentiate between diseases associated with this histologic change

Hammer and Greenson Arch Pathol Lab Med 2013 137: 1216-9
NRCD

Review diagnosis – review biopsy, serology and HLA status

Not CD on review

Diagnosis confirmed

Review dietary adherence

Not adherent, consult dietician

Adherent

Exclude Giardia/pathogens
Microscopic colitis
Exocrine pancreatic insufficiency
Hyperthyroidism
SiBO

Repeat upper GI endoscopy, biopsy, aspirate
Colonoscopy and biopsy
Stool culture
Faecal elastase
Thyroid function

Consider wheat free gluten free diet
Fructose intolerance
Lactose intolerance
Consider FODMAPs

Check histology status, exclude RCD I/II/lymphoma if biopsy confirms
→ RCD
Refractory Coeliac Disease

Failure to heal after 6-12 months of strict GFD
Need to exclude malignancy

RCD1
- Normal immunophenotype: CD3+, CD8+ (>50%)
- Usually responds to GFD, with nutritional support +/- corticosteroids

RCD2
- Aberrant immunophenotype: CD3+, CD8+ (<50%)
- Incomplete response to GFD
- Poor prognosis
- May progress to ulcerative jejunitis and EATL
- Test for T cell clonality
- Continue GFD and nutritional support; anti-inflammatories
Reasons for proceeding with a biopsy

- Antibodies not 100% positive predictive
- Patients take reassurance in a histological diagnosis
- Patients with either IBS or Crohn's disease of the small bowel can be *pseudo-improved* by having GFD
- Baseline histology allows assessment of severity (degree of villous atrophy) for future biopsies
- Some centres will not prescribe GFD unless the CD diagnosis is proven
- CD diagnosis has implications for family members as up to 10% of first degree relatives are affected
- Many patients need an upper GI endoscopy anyway as they have anaemia/other significant symptoms.
- A gastroscopy is more easily tolerated and does not require GA

*Kurien et al Gut 2015; 64:1003-1004*
F62
Presented with painless obstructive jaundice; imaging showed a mass in the head of pancreas; EUS FNA cytology confirmed adenocarcinoma.

Past medical history of Dukes’ A sigmoid carcinoma 8 years previously; hepatectomy for colorectal carcinoma 5 years previously.

Whipple’s pancreaticoduodenectomy performed
Diagnosis

Metastatic colorectal carcinoma to head of pancreas

DD

Intestinal type adenocarcinoma, arising from an intraductal papillary mucinous neoplasm

Pancreatic ductal adenocarcinoma with intestinal phenotype

IHC

CK20 +, CDX-2 +, CK7-, Ca 19-9-
Discussion

Metastatic malignancy involving pancreas is an unusual phenomenon

Increasingly recognised in high volume surgical centres

Resection may be indicated depending on resectability and patient performance status

Effectiveness of resection is dependent on tumour biology of primary tumour

Renal cell carcinoma is the most common source, followed by colorectal carcinoma, melanoma, sarcoma and lung cancer
Discussion

Pancreatic metastases are asymptomatic in more than 50% of cases.

May be picked up on routine oncology follow up.

Symptoms may be non-specific (abdominal pain, weight loss and nausea); rarely, jaundice and GI bleeding.

Suspicion should arise from clinical history, tumour markers and imaging appearances.

Renal carcinoma typically contrast-enhanced hypervascular appearance.

Exclude other lesions by whole body CT or PET scans.

Often a long interval from primary diagnosis.
Discussion

Combined autopsy (n=4995) and resection (n= 973) series suggests only 1.6% of autopsies show metastases to pancreas and in 3.9% of resection specimens

Adsay et al Virchows Archiv 2004; 444:527-535

Direct involvement of pancreas common from gastric or bile duct carcinomas

Metastatic mechanisms for more distal metastases include lymphatic spread, haematogenous dissemination and peritoneal carcinomatosis
Discussion

RCC most common malignancy to metastasise to pancreas

5 year actuarial survival post-resection of 72.6% in 321 patients (DFS 57%)


Comparative survival data for lung resections for RCC mets is 31-44%; may suggest that pancreatic mets are a less aggressive variant

Individual prognostic features in primary tumour not established

Consider alternative treatment options if surgery not possible: immunoreactive cytokines and anti-angiogenic agents
Discussion

Colorectal carcinoma is second most common tumour source

Liver and lung are still the most usual metastatic sites and should be investigated as part of initial work up

Limited data on effectiveness for pancreatic colorectal metastectomy

5 year survival rate of 30% reported (similar to hepatectomy for CRCa mets)


An Italian series found 18/546 (3.2%) resections were for metastatic disease with half being of colorectal origin; median survival 16.5/12 (8-105/12)

Sperti et al Dis Colon Rectum 2009; 52: 1154-1159

Other options should be considered, especially with knowledge of Kras, Nras, MMR and other biological data
Discussion

Melanoma metastases always correlate with poor prognosis

Very little data specific to pancreas: median survival post resection 14/12 (Reddy and Wolfgang Lancet Oncol 2009)

Sarcoma: apart from GIST most are unresponsive to chemotherapy

Encouraging results from isolated hepatic and lung resections; pancreatic resections could be considered but data extremely limited: 5 year survival of 14% is less than for lung or liver resection (Reddy and Wolfgang Lancet Oncol 2009)

Lung cancer rarely goes to pancreas (more commonly bone, liver, adrenals)

Often small cell lung carcinoma, usually treated with best supportive care or systemic therapy

Single case reports of successful resections of NSCLC but most are associated with frequent disease relapse and poor survival rates
Conclusion

Relatively new clinical entity as surgical pathology specimens

Remember possibility of metastasis: clinical history, tumour markers, imaging

Reasonable success with renal cell carcinoma

Colorectal carcinoma is second commonest source of potentially resectable pancreatic metastases

May be seen more frequently in future as criteria for resectability are refined

Bibliography

Adsay et al Virchows Archiv 2004; 444:527-535
Sperti et al Dis Colon Rectum 2009; 52: 1154-1159
Zerbi and Pecorelli World J Gastroenterol 2010; 2: 255-259
Sarajevo November 2016

M24
Subtotal colectomy for inflammatory bowel disease, following failure of medical management

Received a stump of terminal ileum with appendix 8cm and colon 55cm. Macroscopically normal mucosa in distal 13cm with continuous abnormal mucosa in proximal part of specimen with ulceration and pseudopolypsosis
Sarajevo November 2016

M24
Subtotal colectomy for inflammatory bowel disease, following failure of medical management

Diagnosis
Inflammatory bowel disease, unclassified (IBDU)
Features suggestive of indeterminate colitis at Crohn’s disease end of spectrum
Categorisation of inflammation in biopsies taken for the initial diagnosis of inflammatory bowel disease.

Roger M Feakins J Clin Pathol doi:10.1136/jclinpath-2013-201885
Overlapping features

**UC with CD features**
- Fulminant UC
- Segmental disease (caecal patch, response to rx and healing)
- Rectal sparing
- Granulomas (crypt related, infective)
- Ileal disease
- UGI involvement

**CD with UC features**
- Mucosal disease only
- Rectal disease only
- Diffuse colonic disease
A cryptolytic granuloma resulting from rupture of a crypt abscess.
Causes of uncertainty in IBD

- Fulminant (severe/toxic) colitis
- Insufficient information (clinical, radiological, endoscopic, pathological)
- Failure to use major diagnostic criteria
- Failure to recognise pathological variants
- Failure to recognise non-IBD mimics and superimposed conditions
- Attempt to distinguish UC from CD on bx
- Do not attempt to change diagnosis on pouch or diversion specimens
Ileal disease in UC

- Bowel preparation
- NSAIDs
- Backwash ileitis
- Infections
- Bacterial overgrowth
- Ischaemia
- Rare primary UC
Causes to change diagnosis between UC and CD

- CMV
- Pseudomembranous colitis
- Ischaemia
- Radiation
- Drugs
- Microscopic colitis
Mimics of IBD

- Ischaemia
- Radiation
- Diverticular disease associated colitis (DDAC)
- Infections (Yersinia, TB, LGV)
- Diversion colitis/proctitis
- Drugs (NSAIDs, ipilimumab)
- Vasculitis (Behçet’s disease)
Important features

**Clinical:** FHx, PSC, types of S&S, serology, previous surgery, perianal disease

**Radiological:** segmental vs diffuse, SI involvement, strictures, fistulae, wall thickening

**Endoscopic:** type and appearance of ulcers, distribution of disease, ileal involvement

**Pathological:** Prior biopsies and resections
Long-term recurrence in Crohn's disease after IAA

<table>
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<th>Patients (n)</th>
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*From Trigui et al. J Visc Surg 2014; 151: 281-288*