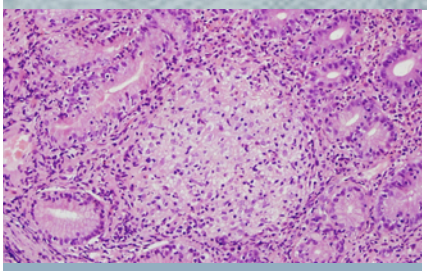
GASTROINTESTINAL PATHOLOGY PROFESSOR N A SHEPHERD



GASTROINTESTINAL PATHOLOGY

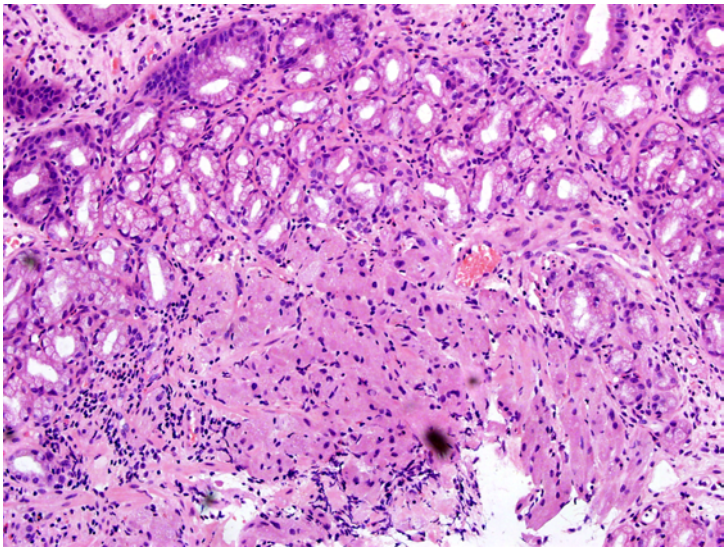
PROFESSOR N A SHEPHERD

Advanced Histopathology Course, East of England Deanery September 2009

1. 09/5199. 86F. Haematemesis. Oesophageal biopsies.

Diagnosis: HSV oesophagitis.

The classical viral cytopathic effects are seen in these oesophageal biopsies. There are intranuclear inclusions and there is the characteristic multinucleation. HSV oesophagitis is most often seen in the immunocompromised but it is occasionally seen, particularly in the elderly, in the immunocompetent. Often, the viral cytopathic effects are focal and the epithelial hyperplasia that accompanies it may mimic squamous dysplasia. There is no question of squamous dysplasia in this case.



2. 09/11822. 59F. Dysphagia. Small polyp at OGJ. Oesophageal biopsies.

Diagnosis: GCT of oesophagus.

These biopsies show the characteristic changes of the OG junction. In addition, there is evidence of a spindle cell tumour. This shows the characteristic granular cytoplasm of granular cell tumours. These tumours are positive for S100 but they are also notably positive for CD68. Only very, very, very occasional cases of malignant GCTs are described in the gastro-intestinal tract and these tumours are usually small and benign.

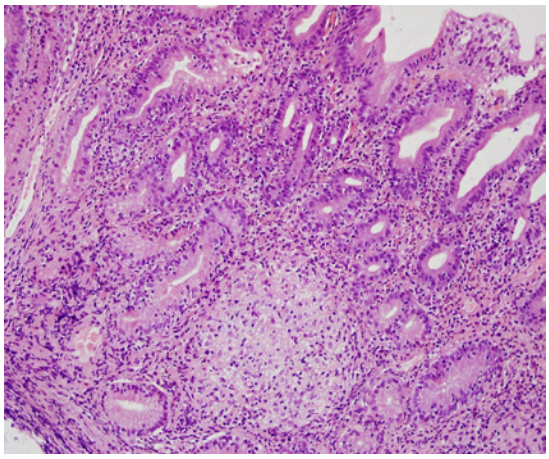
3. 09/10061. 54M. Nodule removed by EMR in long segment Barrett's oesophagus.

Diagnosis: EMR of low grade dysplasia in Barrett's oesophagus.

EMRs are increasing used as a diagnostic and therapeutic procedure in Barrett's oesophagus. In this case, there is non-dysplastic Barrett's-type mucosa but much of the central area of the EMR shows characteristic low grade dysplasia. I will discuss ways of assessing margins of excision and other important parameters during the lecture.

4.07/2266. 42M. Mild gastritis and duodenal nodularity at endoscopy. Gastric and duodenal biopsies.

Diagnosis: Gastric and duodenal sarcoidosis.



There are two slides. They show evidence of florid granulomatous gastritis and duodenitis. In western populations, such granulomatous disease would be mostly commonly caused by Crohn's disease. However, sarcoidosis is also a recognised cause and that is the diagnosis in this case. The following represent three important references on granulomatous gastritis and demonstrate how the cause of this condition varies in different parts of the world:

Ectors NL, Dixon MF, Geboes K, Rutgeerts PJ, Desmet VJ, Vantrappen GR. Granulomatous gastritis: a morphological and diagnostic approach. Histopathology 1993; 23: 55-61.

Maeng L, Lee A, Choi K, Kang CS, Kim KM. Granulomatous gastritis: a clinico-pathological analysis of 18 cases. Am J Surg Pathol 2004; 28: 941-945.

Shapiro JL, Goldblum JR, Petras RE. A clinico-pathologic study of 42 patients with granulomatous gastritis: is there really an idiopathic granulomatous gastritis? Am J Surg Pathol 1996; 20: 462-470.

5.09/6807.49M. Recurrent iron deficiency anaemia. Large rolling hiatus hernia with severe ulceration. Gastric biopsies.

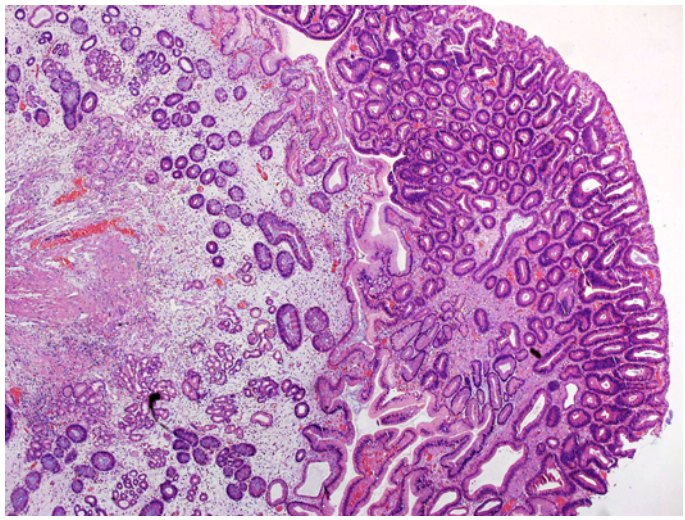
Diagnosis: Iron tablet erosions.

These gastric biopsies show the features of a florid reactive gastritis. In addition, there is evidence of discolouration of the surface, where there is erosion, and these are the characteristic features of iron tablet erosions. A pathologist can confidently indicate that the erosions are caused by iron tablets when one sees this iron encrustation of the surface and the iron deposition within the superficial glands of the stomach.

6. 09/3944. 87F. EMR of gastric body polyp.

Diagnosis: Tubular adenoma arising in chronic atrophic gastritis.

This EMR demonstrates a classical tubular adenoma of the stomach. Note that there is focal intestinalisation within the adenoma. Furthermore, it is important to note the adjacent epithelium. This shows atrophic changes and intestinal metaplasia. This adenoma is arising in a basis of chronic atrophic gastritis. There is no evidence



of malignancy.

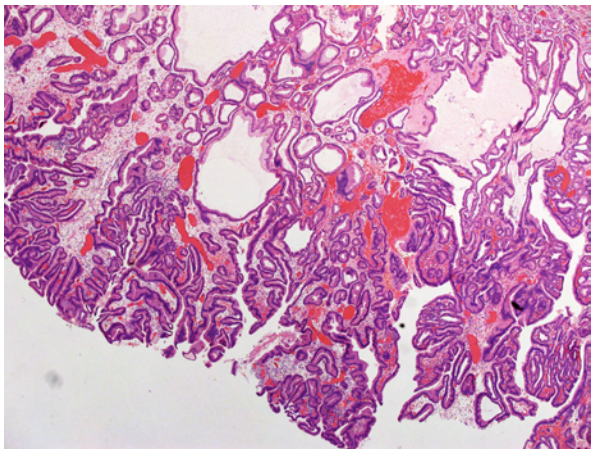
7. 09/3795. 68F. Anaemia and melaena. At endoscopy ulcer crater in mid lesser curve. Gastric biopsies.

Diagnosis: Low grade primary gastric lymphoma.

These gastric biopsies are starting to cut out in some of the sections you have seen. Nevertheless, there is evidence of a focal lymphoid infiltrate and the discussion is around the differential diagnosis of HP gastritis versus primary gastric lymphoma. In this case, in some of the biopsies, there were unequivocal lympho-epithelial lesions (LELs) and the endoscopic features were those of neoplasia. This is a case of primary low grade gastric lymphoma. CK immunohistochemistry is often the most useful tool in the pathologist's armamentarium: this shows the LELs nicely.

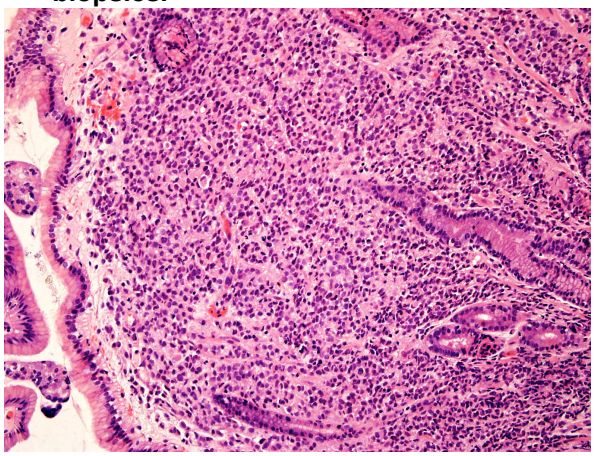
8. 09/11632. 57F. 135mm gastric polyp excised by gastrotomy and linear stapler.

Diagnosis: Foveolar (type 2) adenoma.



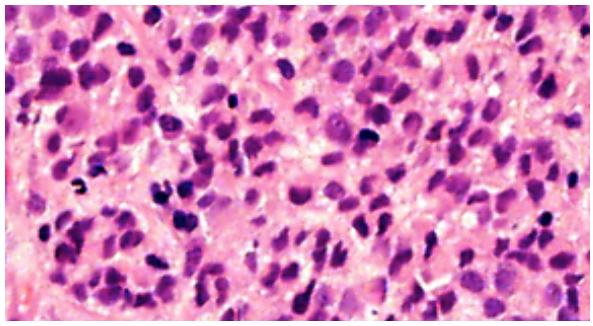
This is a diagnosis that is often missed by pathologists. I think nobody would doubt that, when analysed critically, there is evidence of dysplasia here, albeit low grade dysplasia. These adenomas are often large and tubulo-papillary. They usually show low grade dysplasia throughout. The differential diagnosis rests primarily between foveolar adenoma and a hyperplastic-type polyp. One should note the ratio of glands to stroma and the fact that there is unequivocal dysplastic change. It should also be appreciated that the epithelium is foveolar in type throughout.

9. 09/11909. 83F. Reflux and weight loss. Antral constriction at endoscopy. Antral biopsies.



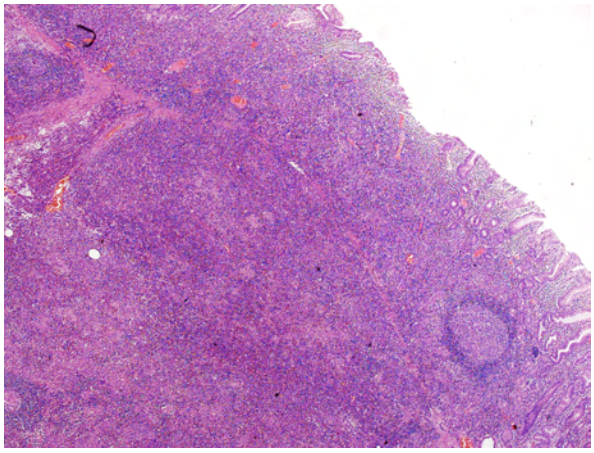
Diagnosis: Metastatic lobular carcinoma of the breast.

These multiple gastric biopsies show a focal infiltrate. On morphological examination, the differential diagnosis is relatively wide and might include endocrine tumours, metastatic malignant melanoma, lymphoma and, perhaps most likely, diffuse-type adenocarcinoma. In fact, when the cells are analysed critically, they show the characteristic features of lobular carcinoma of the breast. This woman had a history of that disease and this is a case of metastatic lobular carcinoma of the breast to the stomach. Perhaps the small bowel is the commonest site for such metastatic disease but gastric and colonic metastases are well described. ER immunohistochemistry was enlightening.



10. 08/18517. 72m. Tumour posterior wall mid body. No pre-operative chemotherapy. Gastric resection.

Diagnosis: Lympho-epithelioma-like carcinoma of stomach.



Initial impressions might suggest that this is lymphoma, perhaps particularly with the prominent lymphoid aggregates. However, one can see that there is an epithelioid element with trabeculae of eosinophilic tumour cells admixed with the lymphoid infiltrate. This is a not uncommon tumour of the stomach, is strongly related to EBV infection, and has a slightly better prognosis than standard adenocarcinoma of the stomach.

11. 08/7154. 74M. Gastric inlet obstruction. Total gastrectomy revealed numerous tumour masses in body of stomach.

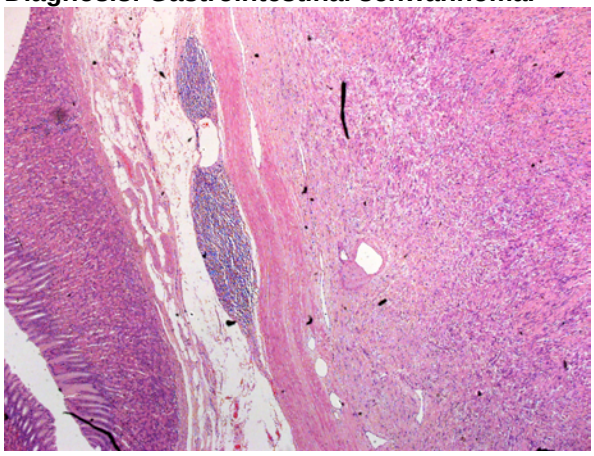
Diagnosis: Endocrine cell hyperplasia, dysplasia and tumours with composite tumour arising in chronic atrophic gastritis.

These two sections of stomach show the spectrum of pathology that one sees, in endocrine cells, in chronic atrophic gastritis. Certainly the latter diagnosis is confirmed in the background. There is then evidence of endocrine cell hyperplasia, endocrine cell dysplasia, endocrine cell "micro-carcinoids" and finally a fully-fledged endocrine cell tumour involving the submucosa. The latter is circumscribed and relatively small. Viewing the sections here, one would suggest that this endocrine cell pathology remains relatively benign.

Nevertheless, there is one feature that should promote concern. In the tumour, there is evidence of glandular differentiation and mucin secretion. This suggests that this tumour may be a composite tumour and have adenocarcinomatous elements. In fact, elsewhere was a large ulcerating adenocarcinoma of the stomach with very adverse prognostic features and there are also several more composite tumours. A somewhat unusual case.

12. 08/2598. 63F. Well circumscribed gastric tumour 30mm in diameter.

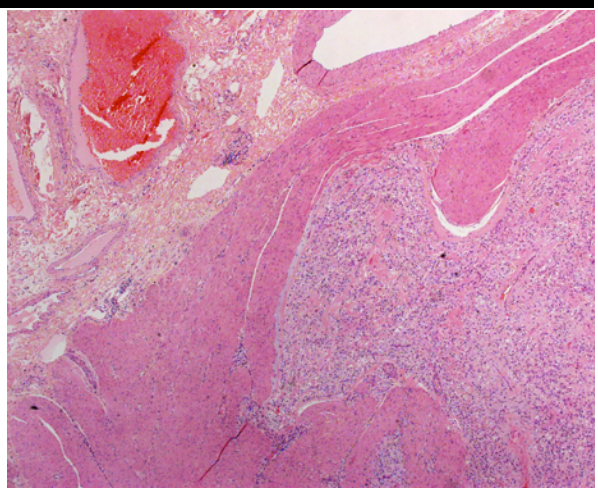
Diagnosis: Gastrointestinal schwannoma.



This well circumscribed gastric tumour is notably spindle-celled. Whilst, usually, one's first thought is of GIST, one should always be careful, particularly when one sees a cuff of lymphoid tissue, as in this case. When one analyses the tumour itself, it shows the characteristic histological features of "ancient schwannoma". This is an entirely benign tumour, strongly positive for S100, and should not be confused with GIST.

13. 08/15148. 74F. 126mm partly cystic mass in stomach.

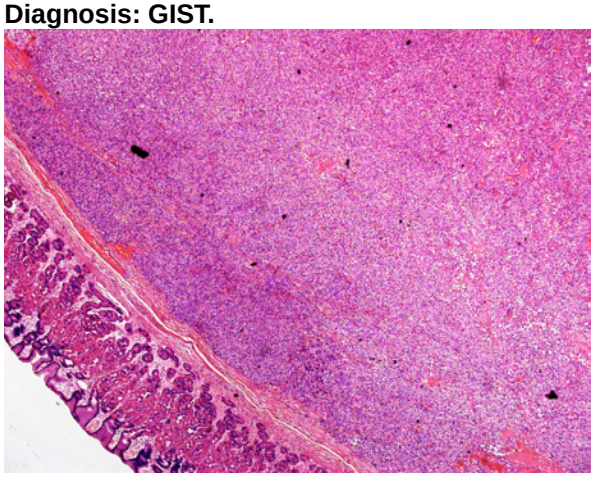
Diagnosis: Telangiectatic GIST.



These sections show strikingly a epithelioid tumour with notably ectactic blood vessels. On a morphological basis, the differential diagnosis rests between GIST, glomus tumour and other primary vascular tumours. In fact, the tumour is strongly positive for CD117 and DOG-1. This case serves to emphasise how variable GISTs are in their morphological appearances.

14. 09/9792. 36M. 39mm mass lesion in fundus of stomach.

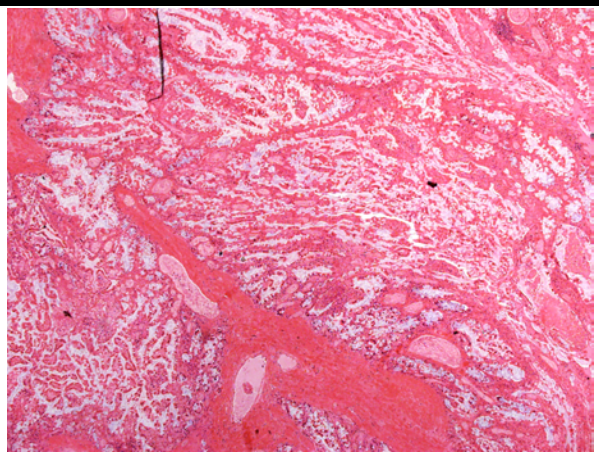
Diagnosis: GIST.



To emphasise the point made above, this tumour is also a GIST. It shows highly variable morphology, some strikingly epithelioid, other areas spindle-celled and still further areas notably pleomorphic.

15. 06/18118. 18F. Right hemicolectomy for intussusception. This section is taken from the ileum at the head of the intussusception.

Diagnosis: Peutz-Jeghers polyp.



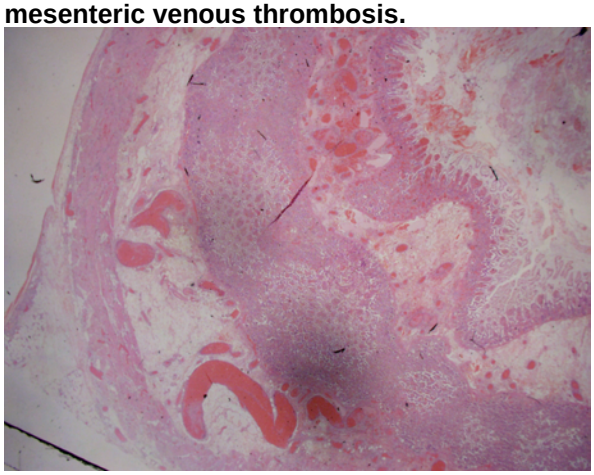
Diagnosis: Peutz-Jeghers polyp

Pathologists may have a problem with intussuscepted tumours in that they often undergo complete necrosis. In this case, whilst necrotic, one can see well the structure of the polypoid lesion and the epithelium and stromal elements

within it. There is the classic arborising morphology of Peutz-Jeghers polyp. This patient was known to have Peutz-Jeghers syndrome.

16. 07/3238. 36M. Polycythaemia rubra vera. Acute abdomen. Small bowel resection.

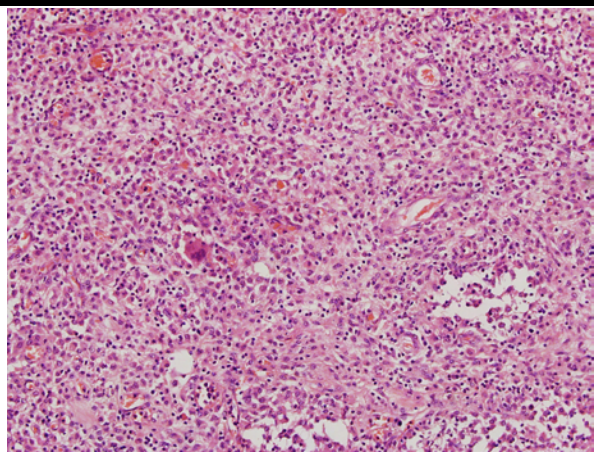
Diagnosis: Ischaemic change due to mesenteric venous thrombosis.



This small bowel resection shows the characteristic histological features of “venous infarction”. There is marked telangiectasia and there is oedematous expansion of the submucosa. Blood vessels are notably dilated. There is some focal thrombosis within veins and this patient, with polycythaemia rubra vera, suffered small bowel infarction because of mesenteric venous thrombosis.

17. 07/53445. 22M. Recurrent abdominal sepsis with intermittent obstruction. Inflammatory mass in right iliac fossa.

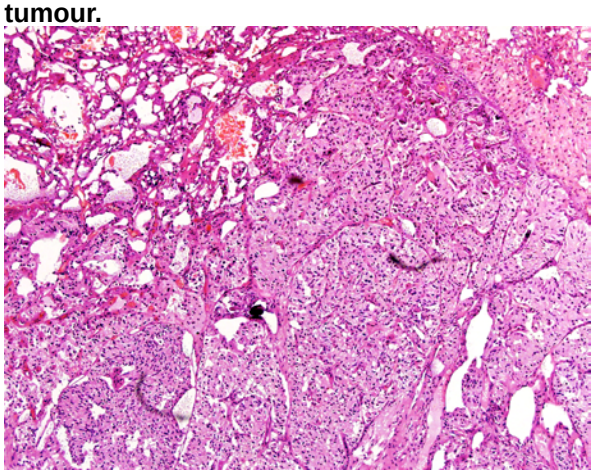
Diagnosis: Yersiniosis.



The sections show a necrotising granulomatous pathology with numerous giant cells. The morphology of the necrotising granulomas is notably geographic. The differential diagnosis includes Crohn’s disease, tuberculosis and various other infective granulomatoses but, in a young patient, the most likely diagnosis is yersiniosis. This diagnosis is confirmed by serological investigation.

18. 08/56543. 52F. EMR of peripancreatic nodule. Metastases in liver.

Diagnosis: Duodenal glandular endocrine tumour.

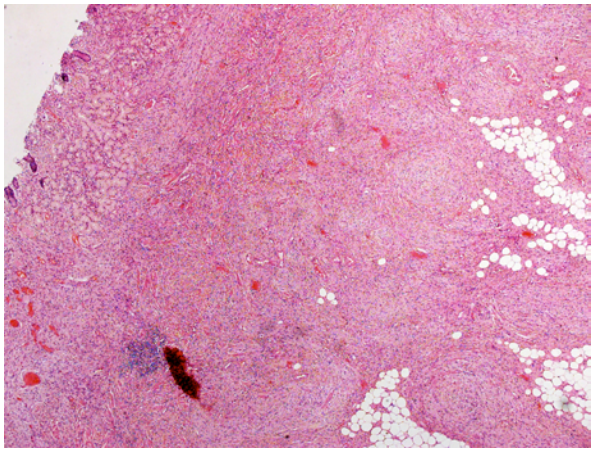


The block is actually from the metastatic disease in the liver. This shows considerable artefact but it does reveal a glandular tumour with prominent psammoma bodies. These are the characteristic features of “somastatinoma” or duodenal glandular endocrine tumour. There is a very important issue with this disease. Even when metastatic, the prognosis is still relatively good and this disease should not be mistaken for psammoma body-producing adenocarcinomas. In the absence of the bodies, these can be mistaken for prostatic or pancreatic adenocarcinoma.

Griffiths DF, et al. Glandular duodenal carcinoid – a somatostatin rich tumour with neuroendocrine associations. J Clin Pathol 1984; 37: 163-9.

19. 10432/08. 59F. Whipple's resection for obstructing tumour of 4th part of duodenum.

Diagnosis: Neurofibromatosis with plexiform neurofibromatosis, ganglioneuromatosis and GIST.

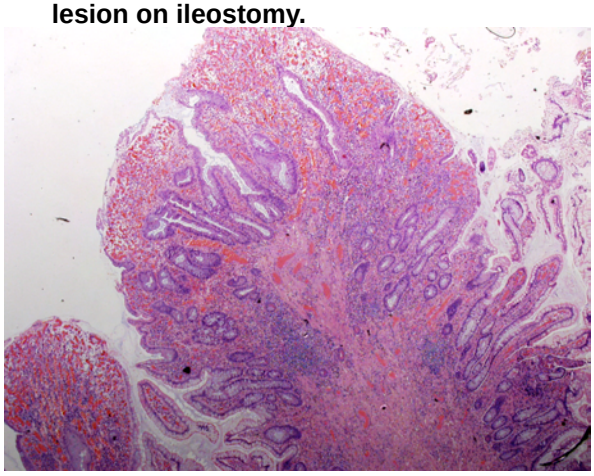


It is relatively easy to spot the ganglioneuromatosis and the plexiform neurofibromatosis in this block. Sadly, in the section I received from the course, the GIST has started to cut out but it can still just about be made out within the muscularis propria.

There are many gastro-intestinal complications in neurofibromatosis and three are represented in this single block.

Fuller CE, Williams GT. Gastrointestinal manifestations of type 1 neurofibromatosis (von Recklinghausen's disease). Histopathology 1991; 19: 1-11.

20. 09/11646. 51M. Panproctocolectomy in 1998 for FAP. Now excisional biopsy of lesion on ileostomy.



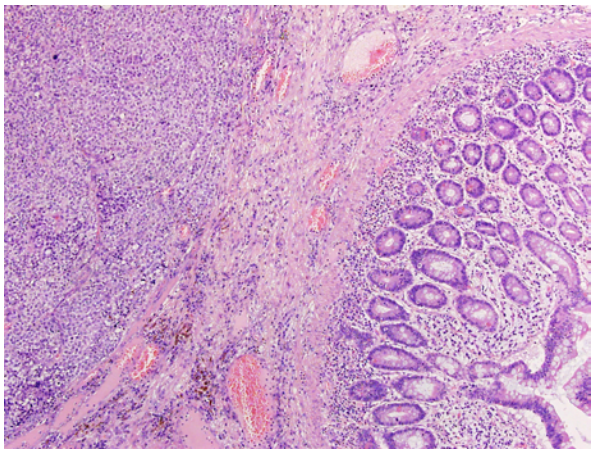
Diagnosis: Ileal adenoma with secondary mucosal prolapse.

I don't think any one can deny that there are changes of mucosal prolapse here. However, some of the epithelium appears notably hyperchromatic. Indeed, there is a small superficial focus that has to be regarded as adenomatous. This is an example of an ileal adenoma (arising in FAP), which has undergone secondary mucosal prolapse at the site

of an ileostomy. We have recently described this phenomenon, whereby there is "secondary" mucosal prolapse due to a pre-existing adenoma at the ano-rectal junction:

Parfitt JR, Shepherd NA. Polypoid mucosal prolapse complicating low rectal adenomas: beware the inflammatory cloacogenic polyp! Histopathology 2008; 53: 91-6.

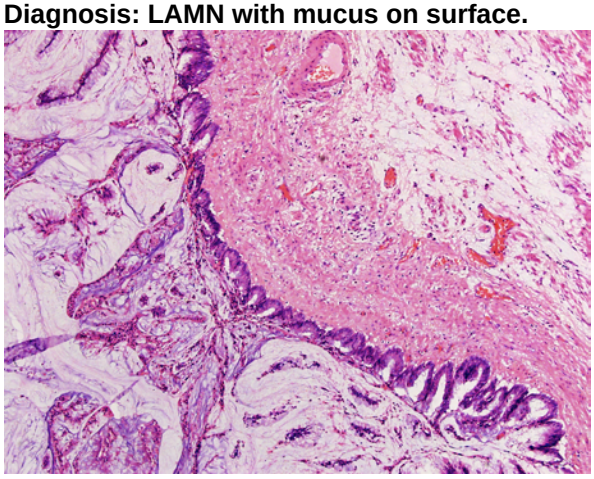
21. 08/12441. 78M. Ileal resection for 56mm diameter single mass.
Diagnosis: Metastatic malignant melanoma.
There is some irony in this case. In initial sections, there was little or no pigment in this single ulcerating tumour of the small bowel. The sections I have seen from much deeper in the block show much pigment and it is clear that this is metastatic malignant melanoma!! Never forget the potential of metastatic malignant melanoma to arise at odd sites around the body. The small bowel, in particular is a site of particular propensity.



Diagnosis: Metastatic malignant melanoma.

22. 05/15578. 74M. Small bowel obstruction. Two strictures on imaging. At operation perforated mucinous tumour of appendix with pseudomyxoma peritonei.

Diagnosis: LAMN with mucus on surface.



The sections show clear evidence of a mucinous cystadenoma in the mucosal aspect of this appendix. However, there is also evidence of mucus "spillage" within the subserosal tissues. The new classification of such appendiceal tumours is "low grade appendiceal mucinous neoplasm" (LAMN) and "mucinous adenocarcinoma of the appendix" (MACA). Both of these tumours have propensity to cause pseudomyxoma peritonei but the disease is much more aggressive for MACA.

Ronnett BM. Pseudomyxoma peritonei: A rose by any other name. Am J Surg Pathol 2006;30:1483-4.

23. 09/14140. 81M. Altered bowel habit. Patchy inflammation and diverticulosis at colonoscopy.

Diagnosis: Diverticular colitis.

The pathological assessment of this case is all about the distribution of the inflammatory changes. This is a complete colonoscopic series. The proximal biopsies and the most distal biopsy are normal. The two intervening biopsies show what is effectively chronic inflammatory bowel disease. This patient was known to have diverticular disease and these are the characteristic histological appearances of "diverticular colitis".

Ludeman L, Shepherd NA. What is diverticular colitis? Pathology 2002; 34: 568-572.

24. 09/6104. 61F. Chronic diarrhoea. Colonoscopy to the terminal ileum normal.

Diagnosis: Pseudomembranous collagenous colitis.

The intact mucosal biopsies here show the characteristic changes of collagenous colitis. However, there is clearly much inflammation. In original sections, this showed the characteristic volcano lesions of pseudomembranous colitis. In sections I have seen from the course, this is somewhat less pronounced. Nevertheless, the appropriate designation is that above. From the literature, this appears to be a disease much closer to collagenous colitis than pseudomembranous colitis. It appears to be towards the severe end of the spectrum of collagenous colitis.

Yuan S, Reyes V, Bronner MP. Pseudomembranous collagenous colitis. Am J Surg Pathol 2003; 27: 1375-9.

25. 09/7601. 63M. Thymic carcinoma and possible Good's syndrome. Diarrhoea. Colonoscopy normal apart from non-specific oedema.

Diagnosis: Acute GvHD.

These colonoscopic biopsies show notable epithelial hyperplasia and very striking apoptotic activity. There is also modest active inflammation. The differential diagnosis of such prominent apoptotic activity includes primary viral infections (particularly primary HIV infection), a drug effect and acute GvHD. In fact, the latter was the appropriate diagnosis in this case. The severity of the disease is proportional to the apoptotic activity.

26. 09/5042. 76F. PR bleeding and diarrhoea. Diverticular disease throughout colon and pseudo-membranes present.

Diagnosis: Crohn's disease and melanosis coli.

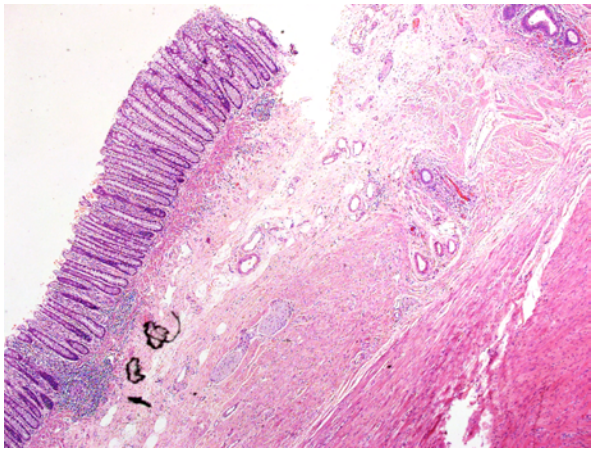
Despite the presence of pseudomembranes, the biopsies show patchy chronic active inflammation and notable melanosis coli. There are also small granulomas. The histopathological features were deemed characteristic of Crohn's disease. Both Crohn's disease and ulcerative colitis, particularly when severe, can mimic pseudomembranous colitis at endoscopy.

27. 09/5158. 29M. Acute diarrhoea and rectal bleeding. Unusual patchy erythema in descending colon and sigmoid colon. Normal above and below.

Diagnosis: Ischaemic colitis.

The changes seen in the slides from the course are subtle but there is evidence of epithelial withering and superficial erosions. There is also the rather characteristic fibrosis in the lamina propria and hyperplasia of the affected epithelium. These are the histological changes of ischaemic colitis. The patient was a smoker and known arteriopath.

28. 09/2135. 42F. CT of colon shows sigmoid cancer. Anterior resection.



Diagnosis: Sigmoid colonic endometriosis.

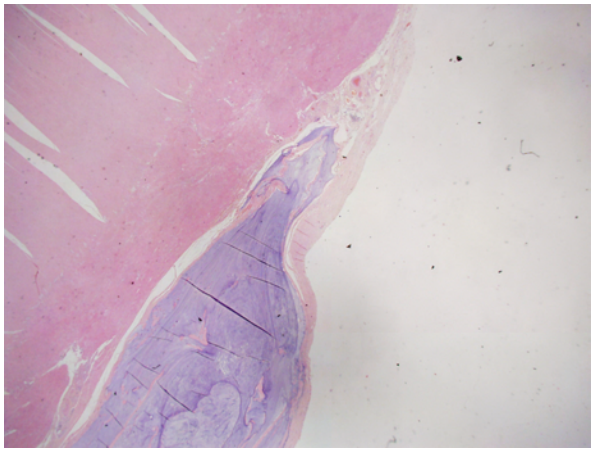
Both clinically and radiologically, this woman was felt to have cancer. However, the slides show classical colonic
endometriosis involving the submucosa, muscularis propria and subserosa. Occasional cases of endometrioid adenocarcinoma complicating colorectal endometriosis are described. Further, endometriosis may cause inflammatory changes in the overlying mucosa, mimicking chronic inflammatory bowel disease.

Gupta J, Shepherd NA. Colorectal mass lesions masquerading as chronic inflammatory bowel disease on mucosal biopsy. Histopathology 2003; 42: 476-481.81.

Petersen VC, Underwood JC, Wells M, Shepherd NA. Primary endometrioid adenocarcinoma of the large intestine arising in colorectal endometriosis. Histopathology 2002; 40: 171-6.

29. 08/17586. 56M. Diverticular sigmoid stricture with pelvic abscess and adherent small bowel loop. Hartmann's resection.

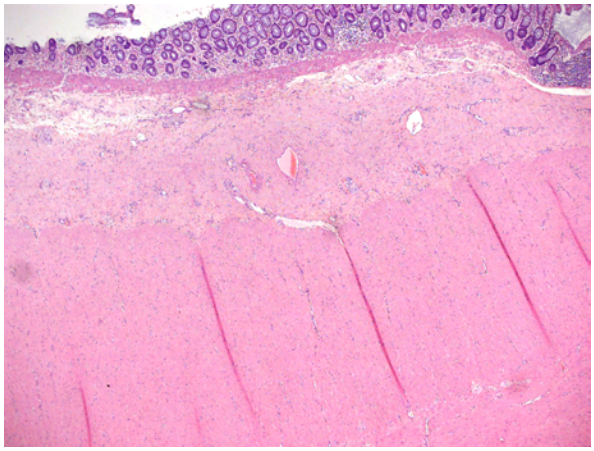
Diagnosis: Mucus pools due to diverticular disease.



When one sees acellular mucin pools, one is always concerned that one is dealing with pseudomyxoma peritonei. However, mucus pools are a recognised complication of diverticulosis, whether they are present in the sigmoid colon or in the appendix. In this case, this small mucinous pool (and it does have some adjacent epithelium in some of your sections) is undoubtedly related to the presence of diverticulosis and does not, necessarily anyway, imply a diagnosis of pseudomyxoma.

30. 07/5651. 34M. Megacolon and sigmoid volvulus. Total colectomy.

Diagnosis: Visceral myopathy.



The diagnosis of visceral myopathy is a highly specialised one. In this case, one can make out subtle abnormalities of the circular muscle with cellular vacuolation and hypercellularity. The changes contrast with the relatively normal longitudinal muscle. In the superficial aspects, there is also some fibrosis. An EVG is a useful stain to do as this disease shows both fibrosis and elastosis. It is admitted, however, that the changes are often subtle.

31. 09/13211. 63M. BCSP. Pedunculated 20mm polyp in rectum marked with tattoo.

Diagnosis: Filiform serrated adenoma.

This is a recently recognised variant of serrated adenoma. Conversely, it appears to be more prominent in the left colon and rectum. These polyps are usually large and show the very characteristic histological appearances seen here.

Yantiss RK, Oh KY, Chen YT, Redston M, Odze RD. Filform serrated adenomas: A clinicopathologic and immunophenotypic study of eighteen cases. Am J Surg Pathol 2007;31: 1238-45.

32. 09/1476. 23F. Total colectomy for colitis failing medical therapy.

Diagnosis: Ulcerative colitis with IV cyclosporin effect mimicking dysplasia.

This section show characteristic changes of ulcerative colitis. However, the mucosa also appears cytologically abnormal. Note that the changes are very diffuse. This is a characteristic complication of IV cyclosporin treatment. This is not dysplasia but merely a mimic of it.

Hyde GM, Jewell DP, Warren BF. Histological changes associated with the use of intravenous cyclosporin in the treatment of severe ulcerative colitis may mimic dysplasia. Colorectal Dis 2002; 4: 455-8.

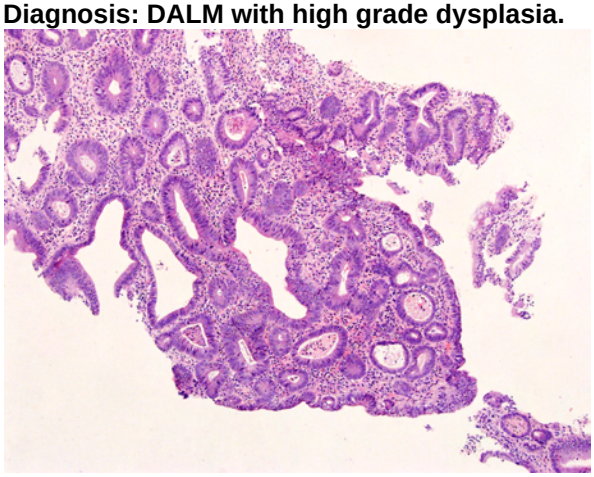
33. 09/85123 (or 09/6645). 58M. Right hemicolectomy for polyp adjacent to ICV.

Diagnosis: Inflammatory polyp with bizarre stromal cells.

This is a simple inflammatory polyp of uncertain cause. Its pathological interest came from biopsies which showed the notable spindle cells in the superficial aspects of the polyp. These are the "bizarre stromal cells" that are more characteristically seen in small polyps at the oesophageal-gastric junction. They are what their name suggests and they do not have any malignant potential.

34. 09/8716. 62M. Longstanding ulcerative colitis. 25mm polypoid lesion in sigmoid colon.

Diagnosis: DALM with high grade dysplasia.



I do not think that any one would deny that there is dysplastic change in this lesion. So the differential diagnosis rests between a sporadic adenoma in a patient with UC and polypoid dysplasia complicating UC. There has been much debate in the literature on how you differentiate these two conditions. I would suggest that "context" is the most important. Does the lesion occur in any area affected by ulcerative colitis? How long has the patient had ulcerative colitis for? Is there dysplasia in flat mucosa away from the lesion? The management of such lesions has changed with the recent acceptance that most dysplastic lesions complicating ulcerative colitis can be removed at endoscopy. Thus the pathological debate can occur after endoscopic removal of the lesion rather than prior to colectomy.

35. 09/8704. 78M. Ca bladder. Sigmoidoscopy shows colitis to the limit of examination.

Diagnosis: Acute radiation colitis.

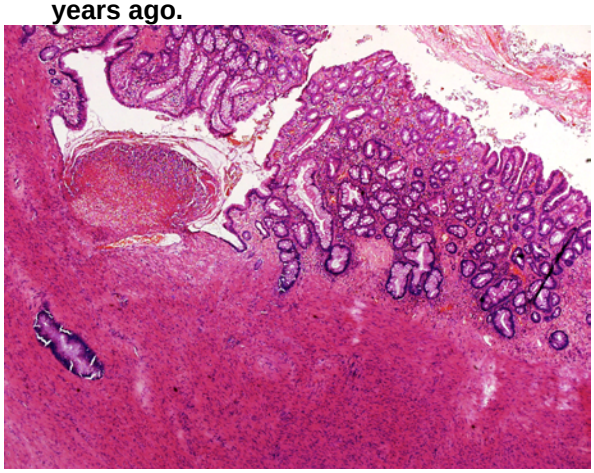
These biopsies show the characteristic changes of acute radiation proctitis. The changes are similar to those seen after short course radiotherapy for cancer of the rectum. In this case, the biopsies were taken early after radiotherapy for cancer of the bladder and the features seen are highly characteristic. Do not mistake these for diffuse dysplasia!

36. 09/10649. 60M. BCSP. 12mm pedunculated polyp in rectum.

Diagnosis: Polyp cancer with all three adverse features.

This polyp shows cancer complicating an adenoma. In this case, the tumour is poorly differentiated, shows vascular invasion and, perhaps in other sections, is present at the resection margin. Further surgery is indicated….

37. 09/1669. 61F. Rectovaginal fistula. Hartman's procedure. Cervical cancer 12 years ago.



Diagnosis: Radiation colitis with colitis cystica profunda and dysplasia.

Radiotherapy is a well recognised cause of the three pathological changes one can see here. The first is proctocolitis cystica profunda. The second is undoubted neoplastic change. Best seen towards one end of the section, there is undoubted "adenomatous" change and in other sections there was convincing evidence of adenocarcinoma. There is also evidence of widespread ulceration and the changes of fistulation can also be demonstrated. This case nicely demonstrates three long term complications of radiotherapy, namely fistulation, proctocolitis cystica profunda and neoplastic change in the colon.

38. 09/4769. 70M. BCSP. 18mm polyp in sigmoid colon.

Diagnosis: This is what we call high grade dysplasia in adenomas!

This sigmoid colonic polyp shows, predominantly, a tubulo-villous adenoma featuring low grade dysplasia. However, there is an additional notable

area where there are marked cytological and architectural changes. This is what we should now regard as, and only as, high grade dysplasia. High grade dysplasia should be limited to cases where, in any other organ, one would regard the changes as those of intramucosal adenocarcinoma. When we do this, particularly as part of BCSP, we will have a clinically useful pathological parameter on which to judge subsequent risk of metachronous cancer.

39. 09/9797. 79F. Right hemicolectomy for obstructing tumour of ascending colon. Two blocks of appendix and ascending colon

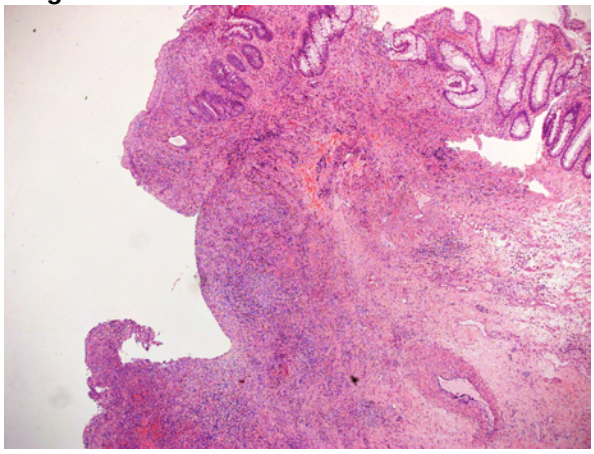
Diagnosis: The serrated neoplasia pathway.

The appendix, in this case, shows the characteristic changes of a "sessile serrated polyp". Note, in places, the transverse orientation of the crypts basally. Almost the whole of the appendix is involved by this lesion. The separate section shows a mucinous tumour of the proximal colon. Critically analysing the epithelium, one can see that this, too, shows serrated features. This is an example of the "serrated neoplasia pathway". Jass believes that this accounts for about 20% of all proximal colorectal cancers. The morphological features of "serrated carcinoma" (described by Jass & Makinen in the following references) should be sought in all cases, but especially in right-sided colon cancer.

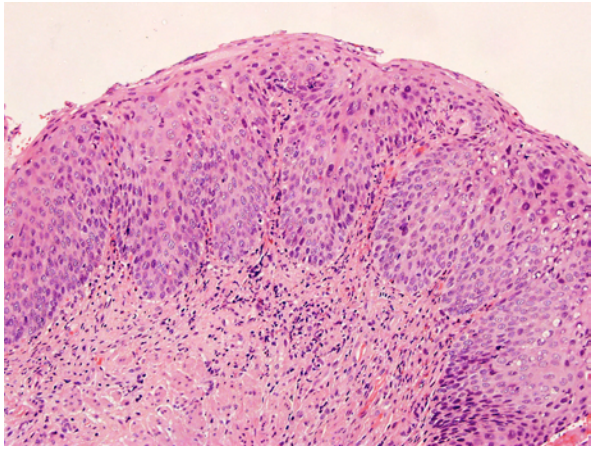
Jass JR. Classification of colorectal cancer based on correlation of clinical, morphological and molecular features. Histopathology 2007; 50: 113-130.

Makinen MJ. Colorectal serrated adenocarcinoma. Histopathology 2007; 50: 131-150.

40. 08/13666. 32M. HIV positive. Ulcer laterally in anal canal and lower rectal mucosa with slightly rolled edge.
Diagnosis: AIN and LGV



There are two separate pathologies here (beloved of FRCPath examiners!!). Firstly, there is obvious evidence of AIN disease here. We can debate the grade!! Secondly, there is evidence of an ulcerating inflammatory pathology. Clinically, this was thought to represent anorectal Crohn's disease. There is certainly evidence of chronic active inflammation and ulceration afflicting the lower rectal mucosa. In fact, ultimately, a diagnosis of LGV proctitis was made. This is now a relatively

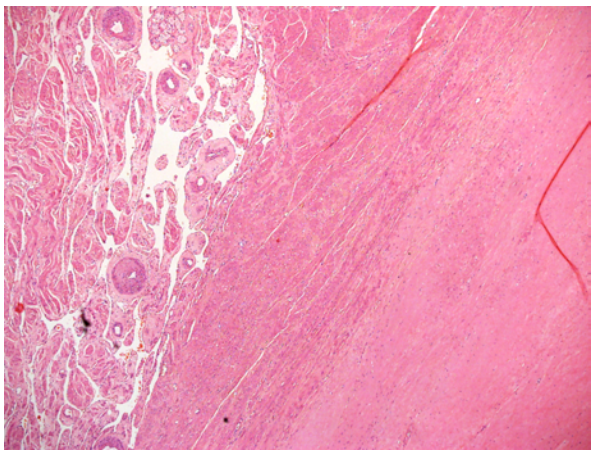


common complication of established HIV/AIDS in homosexuals. It is a strong clinical and pathological mimic of anorectal Crohn's disease.

41. 08/1828. 75M. Recurrent 85mm anorectal mass after previous chemotherapy.

Diagnosis: Glivec-treated GIST.

You may rightly say that there is not too much to see in this tumour. It largely consists of hyalinised tissue containing occasional spindle cells and moderately plentiful blood vessels. There are occasional areas where there is haematoxyphil accumulation, possibly iron as the result of necrosis. In one area in all the slides I have seen, there is a cellular area where spindle cells are notably hyperchromatic. These are the very characteristic features of GIST treated with imatinib. It appears that it is the small cellular areas, which are positive for CD117 and DOG-1,



which are responsible for recurrence of disease after imatinib treatment. It may be that these areas show a second, different, CD117 mutation.

42. 08/30105 (or 08C7081). 77F. Irregular plaque-like anal and perianal change. Wide local excision.

Diagnosis: Paget's disease with invasive adenocarcinoma.

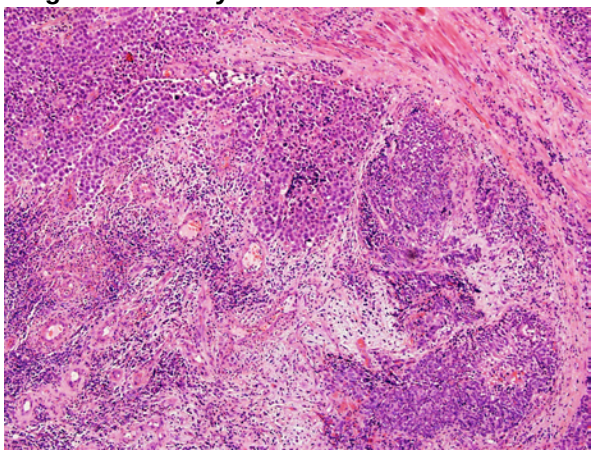
The unusual features seen here are due to florid involvement of the anal squamous epithelium by Paget's disease juxtaposed to the glandular neoplasm that is causing it. Where this squamous epithelium is better orientated, one can see the characteristic features with Pagetoid spread. However, elsewhere, there is a notable glandular tumour which is juxtaposed to the anal canal-type squamous mucosa and produces these unusual histological appearances.

This is anal gland neoplasia complicated by Paget's disease. It is important to understand the Paget's disease may occur for three main reasons: primary perianal Paget's disease, with similar features to those of Paget's disease elsewhere, Paget's disease due to anal canal glandular neoplasia, which may be associated with frankly invasive adenocarcinoma, and finally Paget's disease caused by involvement of the anal epithelium by local spread from a primary rectal carcinoma. Immunohistochemistry can differentiate these three types of Paget's disease and this is clearly very important for further management.

Shepherd NA. Anal intraepithelial neoplasia & other neoplastic precursor lesions of the anal canal and perianal region. In R D Odze (ed) Gastrointestinal Clinics of North America, 2007.

43. 09/14293. 78F. Anal canal ulceration and induration extending into lower rectum. Anal biopsies.

Diagnosis: Poorly differentiated SCC.

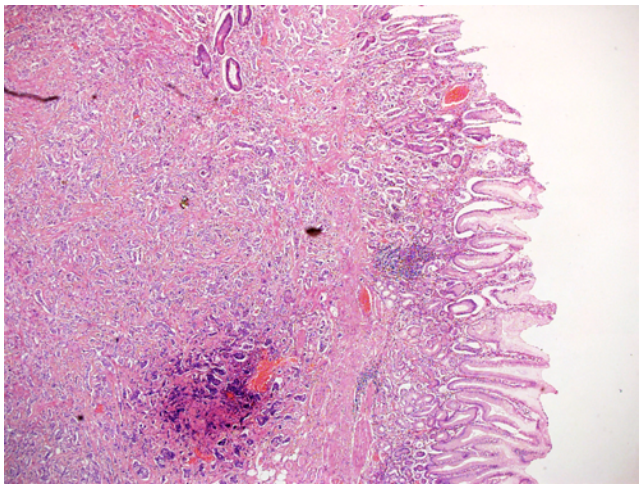


Biopsies of "small blue cell" tumours of the anal region are not so uncommon. It is very important to exclude malignant melanoma and malignant lymphoma, both of which occur as primary tumours at this site. However, in this case, one can see that the tumour is somewhat epithelioid and, in this case, the appropriate diagnosis is very poorly differentiated carcinoma. It was CK5/6 positive and was thus deemed to be a poorly differentiated squamous cell carcinoma.

44. 09/14828. 81F. Rectal bleeding. Indurated ulcer at anorectal junction. Anal biopsies.

Diagnosis: Mucosal prolapse changes.

This is a case where mucosal prolapse at the ano-rectal junction (often called inflammatory cloacogenic polyp) may mimic neoplasia. Particularly when the biopsies are malorientated, the hyperplastic glands, sitting in fibro-muscular connective tissue, can be concerning.



GASTROINTESTINAL PATHOLOGY

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