However, squamous mucosa in the rectum is an abnormal and rare entity. The progression of metaplasia to dysplasia and subsequently to neoplasia is a logical sequence and is well established in the genesis of colorectal, oesophageal and cervical carcinomas. The progression of rectal squamous metaplasia is not established for primary rectal squamous cell carcinoma, although malignant change in rectal squamous metaplasia or leucoplakia has rarely been described.[1]

A 5-month-old boy, born full term by vaginal delivery, presented with abdominal distension and delayed passage of meconium postnatally. Abdominal examination revealed distension. No mass or organomegaly was present; however, a faecolith was palpable. The child was managed conservatively and on follow-up a barium enema was carried out, which showed a transition zone at the rectosigmoid region. The rectosigmoid ratio was reversed and considerable contrast medium was seen in the 24-h film. Hirschprung's disease was clinically diagnosed.

The patient was taken up for surgery at the age of 5 months and Swenson's operation (transanal) was performed. Intra-operatively, a transition zone was seen at the rectosigmoid region with dilatation of the proximal colon. Nearly 8 cm of the proximal dilated bowel was resected and pulled through, followed by anastomosis with the anus. The resected large bowel was subjected to histopathology. Gross examination showed 13 cm of colon, of which the proximal 8 cm was dilated (luminal diameter of 3.5 cm) and the rest of the segment was narrowed (luminal diameter of 1.5 cm).

Microscopic examination of the narrowed part of the segment showed an absence of ganglion cells in the Auerbach's plexus along with neuromatoid and muscular hyperplasia. In addition, a distinct focus of squamous epithelium was noticed, with colonic lining on both sides. No dyplasia was evident in the squamous epithelium (fig 1).

This focus was 7 cm away from the anal-subcutaneous junction. No marked inflammation or other pathology was noted within and in the adjoining areas of the squamous epithelium.

Davis[2] first reported squamous metaplasia of the rectum in 1938. Since then it has been recorded in association with colorectal adenocarcinoma, adenomatous polyp, long-standing ulcerative colitis, anal dermatitis, rectal prolapse and irritable bowel syndrome.[3] It was also noted in an elderly woman who was asymptomatic and who had a family history of colorectal carcinoma.[4]

Histologically, three features establish the diagnosis of rectal squamous metaplasia[4]:

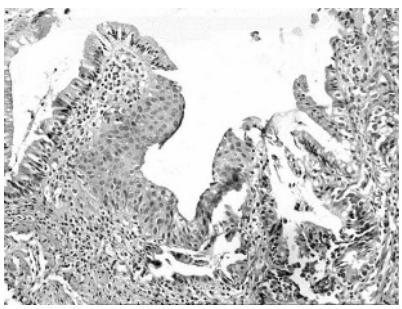

Figure 1 Microphotograph showing squamous epithelium in between the colonic epithelium (haematoxylin and eosin, ×240).

- the absence of primary squamous cell carcinoma elsewhere
- the focus metaplasia is not adjacent to any fistulous tracts
- lack of continuity with the squamous epithelium of the anus.

The index case satisfies all the three criteria. The aetiology hypothesised for squamous metaplasia is ectopic nests of squamous epithelium or, alternatively, stimulation of the uncommitted stem cells seen at the base of normal mucosal crypts on electron microscopy.[5] A similar explanation may hold true in the index case.

The implication and association between squamous metaplasia and Hirschprung's disease is not clearly known and has not been described in the literature to the best of our knowledge. Hence, we conclude that the presence of squamous epithelium in the rectal mucosa is worthy of mention and the remaining mucosa may be examined for the presence of the same. The cause and effect association needs to be further investigated and established.

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Gastric carcinogenesis after long-term observation of clinical course without any treatment in a patient with attenuated familial adenomatous polyposis

We report on a 51-year-old female patient with familial adenomatous polyposis (FAP) that developed into advanced gastric cancer after long-term observation without any treatment (>12 years). We observed gastric carcinogenesis using immunohistochemistry. The patient had a FAP pedigree, and onset of FAP is reported in 11 members of her family. During clinic visits, a total of 12 endoscopies of the large bowel were carried out and tubular adenoma of group 2–3 was seen during all examinations. In a recent detailed examination of the upper gastrointestinal tract, Borr type III gastric cancer was found, with many hepatic metastasis lesions and many polyps in the fundic gland (fig 1A,B).

Systemic chemotherapy was not effective and the patient died on 3 June 2005. A pathological autopsy was carried out with the consent of her family.

Materials and methods

All specimens obtained during autopsy were stored in 10% formalin. Haematoxylin and eosin staining was carried out using a routine technique. Immunohistochemical methods were used on paraffin wax-embedded specimens after formalin fixation. Monoclonal antibodies were used for immunostaining of p53 and cytokeratin 7, the dilution ratios of which were as follows: anti-p53 antibody (1/50, DO-7, Novocastra Laboratories Ltd, Newcastle, UK) and anti-cytokeratin 7 antibody (1/100, OV-TL 12/30, DakoCytomation, Ely, UK). A portion of the specimens was used to perform double staining to detect p53 protein and cytokeratin 7 expressions by using EnVision/AP reagent (Dakocytomation, Code noK4017/4018).

Discussion

Few reports exist on the long-term observation of the natural medical history (without any treatment) of patients with FAP who have an obvious family history. Our patient was extremely rare; she developed concomitant advanced gastric cancer during the clinical course of the disease. Gastric cancer complications in patients with FAP are not highly reported in the literature.[1–5] Most patients, however, had gastric cancer in the gastric antrum and adenoma derived from intestinal metaplastic lesions in the gastric antrum. Because in our patients gastric cancer occurred in the upper gastric body without surrounding adenoma, there is the possibility that a factor other than the generally speculated carcinogenesis from adenoma was associated. Figure 2A shows gastric mucosa in the vicinity of gastric cancer. No chronic gastritis accompanied by atrophy was observed. Almost normal gastric mucosa was seen, with little atrophy or inflammation. When dissection of the mucosa was conducted, however, haematoxylin and eosin staining showed sporadic atypical cells in a few areas (fig 2B). Two atypical lesions displaying low-grade dysplasia were noted at higher magnification (fig 2C). Focusing on this region, sequential sections were prepared for double staining for p53 protein and cytokeratin 7. The region with two protein expressions in atypical cells was found consistent with this area. It was confirmed that these cells express both p53 and cytokeratin 7 (fig 2D). Fundic gland polyps around the cancer tissue were concomitantly subjected to the two immunostaining techniques. Expression of protein was found and it was not considered possible that carcinogenesis resulted from atypia of the fundic gland polyp. Thus, in this case, as a mechanism of gastric carcinogenesis rather than conventional carcinogenesis from adenoma or atypical transformation of fundic gland polyp in the foci of atypical cells in normal gastric mucosa, malignancy was induced via expression of p53 or cytokeratin 7. Adenocarcinoma developed and gradually extended deeper, to grow into a highly malignant adenocarcinoma with poorly differentiated type.

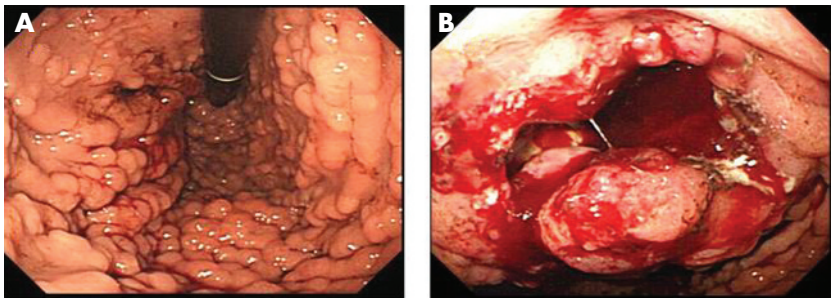

Figure 1 (A) Many polyps in the fundic gland region and (B) Borr type III advanced gastric cancer with deep ulcer in the upper gastric body, observed by endoscopic examination.

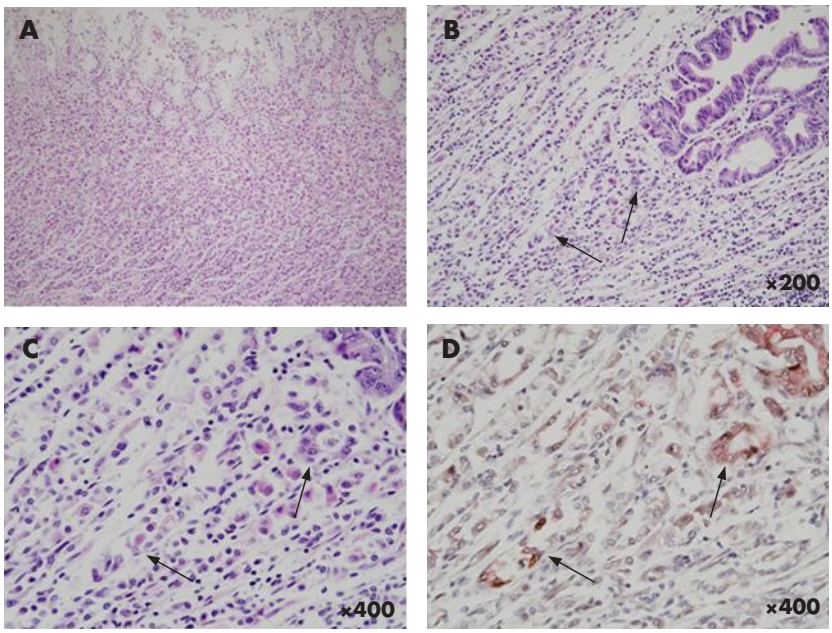

Figure 2 An almost normal gastric mucosa is seen, with little atrophy or inflammatory findings at the gastric mucosa in the vicinity of the gastric cancer (A). Sporadic atypical regions are seen developing in a few areas (arrows) around the gastric carcinogenesis at low power magnification (B) and at higher magnification (C). Double staining of p53 protein and cytokeratin 7 showed apparently positive (arrows), consistent with the region noted to be atypical by haematoxylin and eosin staining (D).

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Intracapsular melanoma: a new pitfall for sentinel lymph node biopsy

Sentinel lymph node biopsy (SLNB) has become an established technique for the staging and treatment of cutaneous melanoma.[1 2] SLNB is very accurate in predicting tumour burden in the remaining regional lymph node basin and is also the most important independent prognostic indicator for recurrence and survival when compared with factors such as tumour thickness and ulceration.[1]

Large retrospective studies have shown that positive sentinel lymph nodes (SLNs) contain metastatic foci of melanoma cells in the subcapsular, sinusoidal or parenchymal regions. The subcapsular region is most commonly associated and up to 86% of metastatic foci become seeded in this area.[3] An important caveat of SLNB, however, is the false positives which result from benign naevic cells present in the capsule (intracapsular) or trabeculae of SLNs.[4] It is the intracapsular location of these cells, as well as differences in immunostaining and atypia, that aids the pathologist in distinguishing benign naevic cells from metastatic foci.[3–5] Indeed, false-positives resulting from benign naevic cells are a cause for concern and require further investigation.

Melanoma metastases in SLNs are detected routinely by using haematoxylin and eosin histology, and S100, human melanoma black (HMB)-45 and Melan-A immunostaining. S100 and Melan-A cannot differentiate between benign naevic cells and melanoma. By contrast, benign naevic cells usually stain negative with HMB-45, thus aiding in their detection.[3 5]

We report a new pitfall for SLNB in a patient with cutaneous melanoma. We observed that metastatic foci of melanoma cells may also seed the intracapsular region of the lymph node (fig 1). These metastatic foci differ considerably from benign naevic cells in that they exhibit marked atypia, mitotic figures, adjacent lymphatic vessels, positive HMB-45 staining (fig 1E) and destruction of the lymph node capsule (fig 2). After SLNB, our patient underwent elective lymph node dissection, which was negative for metastases.

Our observations suggest that an increased level of vigilance is required when examining intracapsular melanocytic cells in SLNs. We must carefully differentiate between benign naevic cells and metastatic foci in this nodal region. Other patient characteristics aside, distinguishing these two intracapsular entities may be pivotal to prognosis and management decisions. As well, incorrectly diagnosing benign naevic cells in which intracapsular metastases are present would constitute a false-negative SLNB and lead to more dismal patient outcomes.

To avoid missing intracapsular metastases, surgeons and pathologists who perform SLNB should be aware of the clinical and pathological pitfalls of the technique. We must ensure complete integrity of SLN architecture when harvesting and sectioning the lymph nodes. This will ensure a thorough examination of common subcapsular melanoma metastases and decrease the likelihood of missing these newly described intracapsular metastatic foci.

Time will tell whether patients with intracapsular metastatic foci have a different prognosis compared with those with metastases in other nodal regions. It is entirely possible that our findings show a new morphological step for melanoma metastases before the seeding of deeper anatomical regions in the SLN.

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